A STUDY OF 50 CASES OF FOCAL SEIZURES WITH CT SCAN CORRELATION

Dissertation submitted in partial fulfilment of regulation

for the award of

M.D. Degree in General Medicine
(Branch I)

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

April 2011

CERTIFICATE

This is to certify that this dissertation titled ***"A STUDY OF 50 CASES OF FOCAL SEIZURES WITH CT SCAN CORRELATION"*** *submitted by* ***Dr.D.K.SIVAKUMAR*** *to the Tamil Nadu Dr. M.G.R. Medical University Chennai, in partial fulfilment of the requirement of the award of M.D. Degree Branch* ***I*** *(GENERAL MEDICINE) is a original research work carried out by him under our direct supervision and guidance.*

Date :

Professor Dr S Veerakesari. M.D.,
Head of the department
Department of Medicine

Date :

Professor Dr.S. USHA. M.D.,
Guide & Professor
Department of Medicine

Date :

Dr.R Vimala, M.D.,
The Dean,
Coimbatore Medical College
Coimbatore - 641 014

DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF 50 CASES OF FOCAL SEIZURES WITH CT SCAN CORRELATION"** was done by me from April 2009 to September 2010 under the guidance and supervision of **Professor Dr. S.USHA. M.D.,**

This dissertation is submitted to The Tamilnadu **Dr.M.G.R**. Medical University, Chennai, towards the partial fulfilment of the requirement for the award of M.D. Degree Examination, Branch-I (General Medicine) to be held in APRIL 2011.

Place: Coimbatore **DR.D.K.SIVAKUMAR**

Date:

ACKNOWLEDGEMENT

It is my proud previlage to express my sincere thanks to **Dr. R.VIMALA, M.D.**, Dean, Coimbatore Medical College and **Dr. MATHIVANAN, M.S.,** Medical Superintendent, for permitting me to utilize the clinical materials of this hospital.

I express my heartfelt thanks and deep gratitude to the Head of the Department of medicine, Professor **Dr. S.VEERAKESARI, MD**., for giving me inspiration Guidance and help in preparing this dissertation

I also thank Professor **Dr. S.USHA, M.D., Dr. NEDUMARAN, M.D., Dr. M.RAVEENDIRAN, M.D.** for their kind help.

I am also very grateful to professor **DR.K. GOVINDARAJAN., MD., D.M.,** Prof. of Neurology for the help rendered in evaluating the study cases.

I also express my thanks to **Dr. T. GEETHA, M.D, Dr. S.AVUDIAPPAN, M.D., Dr. S.SELVAMANI, M.D.** Asst. Professors in the Department of Medicine in giving me full support During my study.

My sincere thanks to all my Assistant professor for their valuable guidance during this study.

A STUDY OF 50 CASES OF FOCAL SEIZURES WITH CT SCAN CORRELATION

CONTENTS

INTRODUCTION

INTRODUCTION

Epilepsy has been important medical problem of Mankind since ancient times. About 3000 years ago a secondarily generalized major seizure was fully described in Akkadian, the oldest written language, written in Mesopatomia (Now Iraq) **(1).** Epileptic seizures continue to cause significant morbidity even in this computer age and our understanding of its etiology, pathophysiology and management is still in its infancy.

Hippocrates first extensively studied epilepsy. His monograph "On the sacred diseases" stated a basic fact about epilepsy- that it originates in the brain. Unfortunately for many centers superstition and ignorance prevailed over rational inquiry. In fact in India, Epilepsy is still considered a mental disorder and legislation continues to reflect this view. Julius Caesar, Alexander the Great and Napoleon Bonaparte are a few of the famous personalities who are said to have suffered from this disorder.

From the patient's point of view, the diagnosis of epilepsy has far-reaching implications in his personal as well as social life. If even leads to unemployment. Hughling Jackson was the first to classify seizures into generalized and focal (partial) in the year 1870. This classification still holds its place in the clinical description of epilepsy and in its management. International league against epilepsy, classifies epileptic seizures broadly into

I - Partial (focal, local) seizures

II - Generalized seizures

III - Unclassified seizure

With sub division under each categories

Partial seizures are those in which in general the first clinical and Electroencephalographic changes indicate initial activation of a system of Neurons limited to part of one cerebral hemisphere.

Many Investigations have suggested that people with partial seizures are more likely to have recurrence than generalized seizures**(2)**

In the evaluation of partial seizure we the physician utilize various tools. First and foremost is the history of illness and then EEG and Neuro imaging. The incidence of structural abnormality in partial seizure is relatively high when compared to generalized seizure, and it is about 79.3% in a study by S. Misra et al, done at Banaras Hindu University, Varanasi**.(3)**

EEG helps us to identify the functional site of epileptogenesis even though the yield is low and also helps us to identify the mirror focus. In the era of epileptic surgery a clinical approach which mixes the skillful history elicitation, EEG, Neuro imaging together us to localize the site of origin of seizure and thereby helps us to have a better cure rate.

This study was intended to identify the correlation between the clinical history, EEG, and CT Scan in the identification of the site of lesion and also to study the incidence of structural lesion in partial seizures and also to identify clues in the clinical history and examination which points towards structural lesion.

AIM OF THE STUDY

AIM OF THE STUDY

1. To study the age and sex distribution of Focal seizures.
2. To study the etiological factors responsible for Focal seizures
3. To study the value of CT Scan of the brain in the diagnosis of the etiology of Focal seizures.
4. To study the correlation between clinical features and CT Scan findings.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Epilepsy is a disease with a long history and lot of myths and stigma to its name.

Broadly seizures are classified into partial, generalized, unclassified by International league against epilepsy 1981. Partial seizure is defined as a one in which in general the first clinical and electroencephalographic changes indicate initial activation of a system of Neurons limited to a part of the cerebral hemisphere. Partial Seizure is further classified as simple partial and complex partial by the presence or absence of impairment in the level of consciousness.

I Simple partial

II Complex partial,

III Partial evolving to generalized seizure

Partial Seizure is further categorized according to the presence of symptoms into

(Source: Modified from Penfield and Jasper)

CLASSIFICATION OF PARTIAL SEIZURES

A. SIMPLE PARTIAL SEIZURES (CONSCIOUSNESS NOT IMPAIRED)

1. WITH Motor Signs

a. Focal Motor without march

b. Focal Motor with March (Jacksonian)

c. Versive

d. Postural

e. Phonatory (Vocalization or arrest of speech)

2. **With Somato – Sensory or special sensory signs**

Simple Hallucination eg. : tingling, light flashes, buzzing

a. Somato Sensory b. Visual

c. Auditory d. Olfactory

e. Gustatory f. Vertiginous

3. With Autonomic symptoms or sings (including epigastric sensation, pallor, sweating, flushing, piloerection and papillary dilation)

4. With psychic symptoms (disturbance of higher cerebral functions)

These symptoms rarely occur without impairment of consciousness and are more commonly experienced as complex partial seizures.

a) Dysphasic

b) Dynamic (e.g. Deja vu)

c) Cognitive (Dreamy states, distortion of time sense)

d) Affective (fear, anger etc)

e) Illusions (e.g. macropsia)

f) Structural hallucinations (e.g. Music, scenes)

B. COMPLEX PARTIAL SEIZURES (With impairment of consciousness,may some time being with simple symptomatology)

1. Simple Partial onset followed by impairment of consciousness

   a. With simple partial seizures (A1-A4) followed by impaired consciousness

   b. With Automatism

2. With Impairment of consciousness at onset

   a. with impairment of consciousness only

   b. with automatism

C. PARTIAL SEIZURES EVOLVING TO SECONDARILY

GENERALIZED SEIZURES (may be generalized tonic–clonic, tonic or clonic)

1. Simple partial seizures (A) evolving to generalized seizures

2. Complex partial seizures (B) evolving to generalized seizures

3. Simple partial seizures (A) evolving to complex partial seizures and then to generalized seizures.

CONTINUOUS SEIZURE TYPE

Generalized status eiplepticus

Generalized tonic-clonic status epilepticus

Clonic status epilepticus

Absence status epilepticus

Tonic status epilepticus

Myoclonic status epilepticus

Focal status epilepticus

Epilepsia Partialis continua of Kojevnikov

Aura continua

Limbic status epilepticus (psychomotor status)

Hemiconvulsive status with hemiparesis

Precipitating stimuli for reflex seizures

Visual stimuli

Flickering light : Color to be specified when possible

Patterns

Other visual stimuli

Thinking Music

Eating Praxis Somatosensory

Proprioceptive Reading

Hot Water Startle

Charcot used to name simple partial seizure a Bravais-Jackson seizure.The incidence of simple partial seizure among epileptic population is calculated to be around 17%**(4).** Partial seizure may run in families. Recent evidence points to linkage of partial epilepsy to chromosome 10q. **(5)** Patients with history of febrile seizure have the chance of developing later seizure with temporal lobe focus in the adult life in about 5%. Most of the time correct history elicitation helps us to get in to the proper diagnosis and thereby effective management. Because proper history first helps us to localize the side of lesion then further probing regarding the type of seizure whether motor, sensory, psychic helps us to localize to a particular lobe. Then if we still go further in to the history the pattern of movements and the site of involvement and type of progression helps us to further localize to particular area of the lobe. But type of seizure may not always necessarily correspond to location of lesion as evidenced by the study conducted

by JMK Moorthy et al**.(6)**

Common seizure patterns and their localization; (7)

Clinical type	Localization
Somatic motor	
Jacksonian (focal motor)	Pre rolandic gyrus
Masticatory, Salivation, speech arrest	Amygdaloid nuclei, opercular
Contra versive	Frontal
Head and eye turning associated with Arm movement or athetoid – dystonic Posture	Supplementary motor cortex
Somatic sensory	
Somato sensory	Post rolandic
Unformed images, lights, pattern	Occipital
Auditory	Heschl's gyrus
Vertiginous	Superior temporal
Olfactory	Mesial temporal
Gustatory	Insula
Visceral autonomic	Insular-Orbital-frontal cortex
Formed hallucination	Temporal Neo cortex or Amygdaloid-hippocampal complex
Dyscognitive experience	Temporal
Affective states	Temporal

(Adopted from ILAE commission Report – **Epilepsia, volume 42, No.6, 2001; 796 – 803, 2001)**

EPILEPSY SYNDROMES AND RELATED CONDITIONS.

Benign familial neonatal seizure

Early myoclonic encephalopathy

Ohtahara syndrome

Migrating partial seizures of infancy

West syndrome

Benign myoclonic epilepsy in infancy

Benign familiar infantile seizures

Benign infantile seizures (Nonfamilial)

Dravet's syndrome

HH syndrome

Myoclonic status in nonprogressive encephalopathies

Benign childhood epilepsy with centrotemporal spikes

Early onset benign childhood occipital epilepsy (Panayiotopoulos type)

Late onset childhood occipital epilepsy (Gastaut type)

Epilepsy with myoclonic absences

Epilepsy with myoclonic – astatic seizures

Lennox – Gastaut syndrome

Landau – Kleffner syndrome (LKS)

Epilepsy with continuous spike and waves during slow wave sleep (other than LKS)

Childhood absence epilepsy

Progressive myoclonus epilepsy

Progressive myoclonus epilepsies

Idiopathic generalized epilepsies with variable phenotypes

- Juvenile absence epilepsy
- Juvenile myoclonic epilepsy
- Epilepsy with generalized tonic-clonic seizures only

Reflex epilepsies

- Idiopathic photosensitive occipital lobe epilepsy
- Other visual sensitive epilepsies

Primary reading epilepsy

Startle epilepsy

Autosomal dominant nocturnal frontal lobe epilepsy

Familial temporal lobe epilepsies

Generalized epilepsies with febrile seizure plus

Familial focal epilepsy with variable foci

Symptomatic (or probably symptomatic) focal epilepsies

Limbic epilepsies

Mesial temporal lobe epilepsy with hippocampal sclerosis

Mesial temporal lobe epilepsy defined by specific etiologies

Other types defined by location and etiology

Neocortical epilepsies

Rasmussen Syndrome

Other types defined by location and etiology

Conditions with epileptic seizures that do not require a diagnosis of epilepsy

Benign neonatal seizures

Febrile seizures

Reflex seizures

Alcohol withdrawal seizures

Drug or other chemically induced seizures

Immediate and early posttraumatic seizures

Single seizures or isolated clusters of seizures

Rarely repeated seizures (Oligoepilepsy)

Based on seizure and electroencephalographic (EEG) characteristics, age, and evidence of brain pathology, a patient with localization – related / Symptomatic epilepsy often can be classified into one of four groups of epilepsy Syndromes, according to the presumed cerebral lobe in which seizures originate :

temporal lobe, frontal lobe, parietal lobe, or occipital lobe. Extensive and sometimes conflicting literature on cerebral localization using clinical and EEG data exists. This chapter contains a summary of features agreed by the Commission on Classification and Terminology of the International League Against Epilepsy and some relatively recently described syndromes.

Epilepsies and epilepsy syndromes with onset at all ages and

Accompanying seizures types, Localization – related / symptomatic (focal, partial) epilepsies

Temporal lobe epilepsy syndromes (SPS, CPS, TCS)

Frontal lobe epilepsy syndromes (SPS, CPS, TCS)

Pareital lobe epilepsy syndromes (SPS, CPS, TCS)

Occipital lobe epilepsy syndromes 9SPS, CPS, TCS)

I. Temporal Lobe Epilepsies:

A. General Characteristics

Simple partial, complex, or secondarily generalized seizures may occur with onset frequently in childhood or young adulthood. Seizures may occur randomly at intervals, or in clusters. Simple partial seizures are characterized by autonomic or psychic symptoms, or both, and by certain sensory phenomena, such as olfactory and auditory illusions or hallucination. The most common sensation is a rising epigastric discomfort.

B. Routine EEG characteristics:

Routine EEGs may shows

a) no abnormality

b) slight or marked asymmetry of the background activity or

c) temporal spikes, sharp waves, or slow waves (unilateral or bilateral) synchronous or asynchronous; may not be confined to temporal areas.)

C) <u>Sub Types:</u>

1. Amygdala – Hippocampal Seizures

Amygdala-hippocampal seizures are the most common form of temporal lobe epilepsy and generally conform to the general description. Seizures are characterized by rising epigastric discomfort, nausea marked autonomic signs, and other symptoms including borborygmi, belching, pallor, fullness of the face, flushing, arrest of respiration, pupil dilation, fear, panic, and olfactory gustatory hallucinations, Scalp EEG often shows unilateral or bilateral spikes most prominent in the anterior temporal leads.

One variant of amygdale - hippocampal seizures is called the mesial temporal lobe epilepsy syndrome. Such patients demonstrate mesial temporal sclerosis on imaging studies. They typically have a strong family history of epilepsy showing an autosomal dominant inheritance with incomplete penetrance. The patient has seizures (often completed) during infancy or childhood. After a silent period lasting 2 to 15 years, unprovoked partial seizures being in late childhood or early adolescence. The seizures are refractory to medical treatment in 20% to 30% of patients.

2. Lateral Temporal Seizures

Lateral temporal seizures are being as simple partial seizure characterized by auditory hallucinations or illusions, dreamy state, visual misperceptions, or

language disorders (dominant- hemisphere focus). These may progress to complex partial seizures if propagation to mesial temporal or extra temporal structures occurs. Lateral temporal seizures usually lack several of the features typical of mesial temporal seizures, including automatisims, controlateral dytonia, Swerving head movements, body shifting, hyperventilation, and postictal cough or sigh. The scalp EEG often shows unilateral or bilateral spikes most prominent in the middle or posterior temporal leads.

A special subtypes termed autosomal dominant lateral temporal epilepsy (also called autosomal dominant nocturnal epilepsy) has been reported. Onset is in the second or third decade of life. The subtype is characterized by rare partial seizures, usually secondarily generalized, arising mostly in sleep, simple partial sensory phenomena of visual (lights, colors, simple figures) or auditory (buzzing or humming) sense may occur. Paroxysmal activity may be seen in the EEG interictally in the temporal or occipital leads. The condition responds to antiepileptic drugs but may require prolonged administration. Genetic analysis has found linkage to chromosome 10q, locus EBN1, gene KCNQ2.

II- Frontal Lobe Epilepsies

A. Clinical Characteristics.

Frontal lobe epilepsies are characterized by simple partial, complex partial or secondarily generalized seizures or combinations of these. Features suggesting frontal lobe epilepsies are

a) Frequent seizures often in sleep

b) Short seizure duration

c) Minimal or no postictal confusion after complex partial seizure

d) Rapid secondary generalization

e) Prominent motor manifestations that are tonic or postural

f) Complex gestural automatisms (may be sexual) at onset;

g) Frequent falling during seizure; and

h) Frequent episodes of status epilepticus

B) EEG Characteristics

The interictal EEG of frontal lobe epilepsy patients may show

a) no abnormality

b) back ground asymmetry and

c) spikes, sharp waves or paroxymal fast activity that can be

Unilateral or bilateral, unilobular or multilobular. Patients whose seizures originate from the dorsolateral convexity tend to have interictal epileptiform abnormalities that localize to the region of seizure onset. Patients whose seizures begin in the medial frontal region tend to have either no epileptiform activity or multifocal epileptic form discharges. Vertex or midline epileptiform discharges also can be seen with medial frontal foci. Frontal foci not infrequently exhibit spikes or sharp waves in the temporal leads.

III PARIETAL LOBE SEIZURES:

A) General characteristics:

Parietal lobe epilepsy syndromes usually are characterized by simple partial and secondarily generalized seizures. Most seizures remain simple and exhibit sensory phenomena. Most frequently, seizures are of the anterior parietal subtype.

B). EEG Characteristics:

Interictal EEGs may show a) Normal results b) focal slowing or c) focal spikes and sharp waves that are unilateral or bilateral synchronous or asynchronous. Slow and sharp activity spreading beyond parietal leads is not common. Vertex or midline epileptic form abnormalities can be seen with somatosensory seizures arising from the mesial surface of the parietal lobe.

IV- OCCIPITAL LOBE SEIZURES

A. General Characteristics:

Occipital lobe seizures are characterized by positive and negative visual phenomena. Positive phenomena include elementary visual hallucinations often described as bright lights or colored lights. Negative phenomena include amaurosis, scotomas, and hemianopia. The visual phenomena usually are

controlateral to the side of the seizure and may remain stationary or move across the field. Persistent (hours) amaurosis can be a postictal phenomenon.

Other occipital seizure manifestations include tonic and clonic eye deviation, head deviation, blinking, a sensation of eye movement, and nystagmoid eye movements. Eye and head movements usually are controlateral to the side of the seizure focus in occipital seizures (this may not be the case for seizures arising in other areas)

B. ECG Characteristics

Surface EEGs most often demonstrate extensive posterior temporal occipital paroxysmal activity. This pattern may be difficult to distinguish from temporal lobe epilepsy of posterior temporal origin.

ETIOLOGY

A cause may be identified only in about 50% children with epilepsy. Epilepsy with an older age of onset is more likely to have an underlying cause, especially if the patient has partial seizures. In a large series of cases of seizures of unknown cause, the brain has been shown to be macroscopically and microscopically normal so that a metabolic or biochemical cause or cause may be reasonably suspected.

COMMON CAUSE OF SEIZURES OF NEW ONSET

A. PRIMARY NEUROLOGIAL DISORDERS

1. Benign febrile convulsions of childhood
2. Idiopathic epilepsy
3. head trauma
4. Stroke of vascular malformation
5. Mass lesions
6. Meningitis or encephalitis

B. SYSTEMIC DISORDERS

1. Hypoglycemia
2. Hyponatremia
3. Hyperosmolar states like hyperosmolar non ketotic coma
4. Hypocalcemia
5. Uremia
6. Hepatic encephalopathy
7. Porphyria
8. Drug overdose
9. Drug withdrawal
10. Global cerebral ischemia
11. Hypertensive encephalopathy
12. Hyperthermia

CAUSE MAINLY RELATED TO PARTIAL SEIZURES

1. CEREBRAL TRAUMA

a. Birth injury

b. Head injury – cerebral contusion and hemorrhage

2. **STRUCTURAL LESIONS**

a. Vascular malformation

b. Aneurysms c. Cerebral tumors

d. Cysts e. Hydrocephalus

3. **INFECTIONS**

a. Meningitis b. Encephalitis

c. Abscess d. Empyema

e. Syphilis f. Tuberculosis

g. HIV h. toxoplasmosis

4. **INFLAMMATION**

a. Sarcoidosis b. Multiple sclerosis

c. Systemic lupus erythematosis

THE MAJOR KNOWN CAUSES OF EPILEPSY ARE:

1. PERINATAL FACTORS

Perinatal asphyxia and birth trauma leading to subarachnoid, subdural or intraventricular hemorrhage are some of the mechanisms that lead to seizures. A

lesion in the hippocampal region known as ammon horn sclerosis may be caused by anoxia sustained during protracted labor. Excessive moulding of the head during prolonged labour may lead to herniation of the medical part of the temporal lobe over the free edge of the tentorium in the New born. This may compress the posterior cerebral artery. Such herniations may spontaneously reduce, but the ischemic area under gliosis (incisural sclerosis) and may give risk to temporal lobe seizures in the later life.

2. HEAD INJURIES

Post – traumatic seizures are of two types:

a. 'Early seizures' occur within 7 days of the head injury

b. 'Late Seizures' occur after the first 7 days.

Late seizures are very common after missile injury which result in penetration of the dura. Seizures occur in nearly half of these patients. Epilepsy is most likely to occur after an injury that results in loss of consciousness and – or post-traumatic amnesia of more than 24 hours and if there is a skull fracture or intra-cranial hematoma or cerebral contusion. Children, however have a higher rate of posttraumatic epilepsy even with minor head injury.

3. BRAIN TUMORS

Both primary and secondary brain tumors can cause epilepsy. Tumors located in the Rolandic areas are more likely to cause seizures. The most common primary tumor that causes seizures is **meningioma.** Tumors are more likely to cause seizures in adults than in children. In adults with partial seizures the chances of detecting a brain tumor may be as high as 30%.

4. INTRA CRANIAL INFECTIONS

Meningitis and encephalitis commonly cause seizures in the acute phase and epilepsy is a common sequel of these infections, especially in children. Neurosyphilis and tuberculosis are other infections causing epilepsy. Parasitic infection like cysticercosis should be considered as a cause in India. Epilepsy may some times occur following inoculation against small pox and pertussis. Seizure can occur when there CNS involvement in AIDS. It is either because of opportunistic infection like toxoplasmosis or neoplasia like lymphoma or as a part of AIDS dementia complex.

5. CEREBROVASULAR DISEASES

Epilepsy occurs in about 10% of patients with cerebral infarction. Epilepsy, especially partial seizures often occurs in patients with cortical venous thrombosis. Vascular malformations cause epilepsy in adulthood. Though the malformations are present from birth, seizures sometimes do not occur until the fifth decade of life.

Cerebrovascular diseases and stroke become increasingly common cause of epilepsy in the later years of life. A community based study of stroke showed an incidence of seizures by one year of 4% of cases with cerebral infraction, in 18% of patients with intra cerebral hemorrhage and in 28% of patients with subarachnoid hemorrhage. Other studies have emphasized that embolic or hemorrhagic strokes carry the highest risk. However, asymptomatic carotid occlusion and cerebral infraction may be found in patients presenting with epilepsy later in life and seizures may also precede a stroke. Overall 16% of acutely precipitated seizures caused by cerebrovascular events and this percentage increases to 40 in the elderly. Epilepsy is also common manifestation of AV malformations. It may occur in upto 40-50% of patients and most commonly occur in those who have had episodes of hemorrhage or who have been treated surgically.

Arteritis can be accompanied by seizures as a part of stroke like syndrome of acute encephalitis. Between 17 to 50% of patients with SLE and

CNS involvement have seizures. Other vasculitis disorders with the CNS involvement can also cause seizures e.g. PAN, Behcet's disease and MCTD, Hypertensive encephalopathy and bacterial endocarditis also can cause seizures.

6. INTRA CRANIAL TUBERCULOMAS

A tuberculoma is a tumor like mass of typical tubercular granulomatous tissue that produces symptoms of space occupying lesion (SOL).

A Higher incidence of intra-cranial tuberculoma has reported from India. Intra cranial tuberculoma are due to hematogenous spread of infection and are caused by human type of tubercle bacilli. The initial lesion is solitary or a cluster of microscopic lesions generally in the subcortical region consisting of a central area of necrosis surrounded by characteristic epitheloid and giant cell reaction. The lesions enlarge as a result of expansion of individual foci that later coalesce. Caseous necrosis, vasodilation and typical tuberculous cellular infiltration along with surrounding edema add to the mass effect.

Macroscopically, a tuberculoma is a well-circumscribed grayish white, firm mass or lobulated mass with a granular surface measuring a few millimeters to 3-4 cm or more in diameter. Less frequently, tuberculoma may occur as a diffuse infiltrating lesion designed as "tuberculoma en-plaque" The periphery of this lesion tends to be quite vascular, resulting in a 'tumor-blush' on angiography.

CLINICAL PICTURE: The disease primarily affects younger subjects. Fever is rarely present. Usual presentation is that of a slowly growing ICSOL. The symptoms and signs are those of raised ICT, focal or generalized seizures and focal neurological deficit either alone or in combination.

The Focal neurological signs are determined by the location of the lesions in the brain. In children, the cerebellum is more frequently affected whereas in adults the common site is the cerebral hemisphere, the commonest site being the fronto parietal region, Nearly 20-30 % of patients have multiple lesions, which can cause confusion in clinical localization.

Lab Diagnosis: ESR is raised in only a small number of patients. CSF examination may reveal a slight to moderate pleocytosis (10-15 cells) and increase in proteins. No reliable immunodiagnostic test is available as yet. Calcification is uncommon, occurring in only 4-6% and X-ray skull is usually non-contributory.

CT scan is of great help in diagnosing tuberculoma. A tuberculoma is seen as a ring enhancing lesion that may be present as a small or large ring, a disc or a uniformly dense nodular lesion. A ring with a central dense dot – 'target sign' raises the suspicion further. Invariably the enhancing lesion is associated with varying degrees of low attenuating edema. There may be multiple lesions of varying sizes.

7. NEUROCYSTICERCOSIS

According to recent literature cysticercosis is the most common cause of symptomatic epilepsy in the world. The disease is cause by the larval form of the pork tapeworm. It is acquired by feco-oral transmission.

T solium is the tapeworm for which man can be the intermediate host harboring the larval form of the worm. They pass through the lungs and then embolise lodging in skeletal muscle, eyes and the CNS. In the CNS the oncospheres may lodge in the gray matter, at the junction of the gray and white matter or in the subarachnoid space. In tissues the embryos develop into encapsulated larval forms called cysticerci which are filled with clear fluid and contain a visible scolex.

The CNS is involved in 90% of cases of human cysticercosis. The presence of single cysticercus in the CNS in unusual. In more than 80% of cases multiple cysts are found. Cerebral lesion typically evolve from an active to a transitional form and then to an inactive form.

Four types of CNS cysts are encountered in cysticercosis. **Parenchymal** cysts, **Meningeal cysts**, **Ventricular cysts** , **Spinal cord cysts** .

The host can tolerate the worm as long as the embryo is alive. It unusually dies 2-6 years after infection and the ensuing disintegration of the parasite eventually decays into grumose or eosinophilic dessicated materials.

The final stage of this process is characterized by the presence of calcified nodule, presumably the result of dystrophic calcification of the necrotic larva.

Seizures are the most common clinical manifestation at all stages of intra parenchymal infestation, although headaches and focal symptoms are common during the active and transitional stages. Meningeal and intraventricular cysts can result in hydrocephalus.

The mechanism of development of seizures in neurocysticercosis is not known. One hypothesis is that the lesion disturbs the microenvironment of the surrounding neurons either by affecting neurotransmitters or by stimulating axonal reorganization in ways that favour excitation or inhibition.

Serological tests may be useful in the diagnosis, but negative results do not rule out cysticerocis. Indeed, when inflammation is absent even the most accurate test the Enzyme-linked Immuno Transfer Blot is negative in 60-80% of patients and probably is more than 80% of cases involving a single lesion. Sensitivity of serological testing decreases considerably late in the course of the diseases.

CT and MRI Scans can reliably pick up lesions of neurocysticercosis and its associated complications like hydrocephalus. Usually there are multiple lesions, occasionally there only a single lesion. All the stages of the diseases can be detected using these imaging modalities

Treatment with the antihelminthic drugs-praziquental or albendazole may be beneficial during the active and transitional stages and may even help control seizures, but this type of treatment is unlikely to be effective when the disease is inactive. When epilepsy is proved to be resulting from a single lesion, seizure control may be effected by complete resection of the lesion.

8. METABOLIC AND SYSTEMIC DISORDERS may be associated with seizures that abate with correction of the underlying abnormality. These patients are not considered to have epilepsy.

1. **Hypoglycemia** can produce seizures especially when serum glucose levels fall to 20-30 mg.

2. **Hyponatremia** may be associated with seizures at serum sodium levels below 120 meq/1 or at a higher level following a rapid decline.

3. **Hyperosmolar** states including both hyperosmolar nonketotic hyperglycemia and hypernatremia may lead to seizures when the plasma osmolality rises above 330 mosm/I.

4. **Hypocalcemia** with serum calcium levels in the range of 4.3 – 9.2 mg/dl can produce seizures with or without tetany.

5. **Uremia** can cause seizures especially when it develops rapidly. Seizures are reported during dialysis (dialysis dysequilibrim) related to the levels of Aluminium used in the dialysate

6. **Hepatic encephalopathy** is sometimes associated with generalized or focal seizures.

7. **Porphyria** is a disorder of Heme biosynthesis that produces both neuropathy and seizures.

8. **Drug overdosage** can exacerebrate epilepsy or can cause seizures in non-epileptics.

9. **Drug withdrawal** especially withdrawal from ethanol, sedatives or anticonvulsants may be accompanied by one or more GTCS that usually occur within 48 hours.

10. **Global cerebral ischemia** may be associated with spontaneous myoclonus, action myoclonus, and partial or generalized seizures.

11. **Hypertensive encephalopathy** can present with GTCS or partial seizures.

12. **Eclampsia** develops seizures or coma. Eclampsia usually occurs in the third trimester, near term, but can occur upto 2 weeks post partum.

13. **Cerebral venous thrombosis** (CVT); Eclampsia and CVT are two conditions prominently associated with seizures during and just after pregnancy. In India 50% of all strokes in women are related to pregnancy and puerperium and 90% of these are due to sinovenous

infarction. Clinical features include raised ICT, altered sensorium, seizures, focal neurological deficts and cranial nerve palsies. Diagnosis can be established by CT /MRI/Angiogrpahy. The most frequent direct sign in CT scan is the **empty delta sign** (triangular rim of contrast surrounding a clot within the superior sagittal sinuses) which is seen in 30% of case. Treatment includes control of infection, cerebral edema and other supportive measures. Heparinisation has been found to be useful in improving the outcome in recent studies.

14. **Hyperthermia** - causes seizures, confusion or coma, shock and renal failure.

15. **Hypomagnesemia** - Serum Magnesium levels less than 1.3 mmo1/1 can cause seizures.

16. **Hypophosphatemia** – tonic clonic seizures occur with serum phosphate levels less than 1 mg/d1.

17. **Thyroid disease** – Seizures occasionally occur in hyperthyroidism. Seizures are more common with hypothyrodism and may occur in about 25% of patients with myxoedema coma.

In an Indian study of 100 consecutive surgical specimen from cases operated for medically refractory CPS, by Radhakrishnan et al showed 58

patients had Ammon horn sclerosis, corpora amylacea deposition in 54 patients, 6 patients had neoplasm, 31 patients had non specific changes. **(8)**

CLINICAL FEATURES

The clinical symptoms and signs of partial seizures (simple and complex) are discussed here.

A. SIMPLE PARTIAL SEIZURES:

Any part of the body may be involved in a focal seizure depnding on the site of epileptic discharge in the motor cortex. It may the form of jacksonian march, speech arrest or partial dysphasia or epileptic pallilalia. Todd's paralysis may follow a focal seizure and lasts for minutes to honour.

PARTIAL SEIZURES WITH AUTONOMIC SYMPTOMS: Rarely partial seizures may manifest by autonomic disturbance such as vomiting, pallor, flushing, sweating and papillary dilatation.

PARTIAL SEIZURES WITH SOMATOSENSORY OR SPECIAL SENSORY SYMPTOMS: These arise from the sensory cortex and may march in a manner akin to motor seizures. Special sensory seizures consist of auditory, gustatory, olfactory (uncinate seizures) and visual phenomena. Vertiginous hallucinations like intense feeling of dizziness can sometimes occur.

B. COMPLEX PARTIAL SEIZURES:

The most common type of complex partial seizures, occurring in over 90% of patients, is that with psychomotor symptomatology. In about half of the patients the seizure is preceded by an aura. Then a phase of motionless total unresponsiveness begins lasting for about 10-15 seconds. Facial grimace and head turning often precede this phase. The head turning has not localizing value.

The second phase is longer lasting, 20-30 second and consists of stereotyped automatic motor activity or **automatisms.** The most common automatisms involve the face and lips. Chewing movements are called **de novo automatisms** and can arise from either internal or external stimuli. The motor components of the seizures occur during the later phase and take the form of automatisms. These include lipsmacking, chewing or swallowing movements, fumbling of the hands or shuffling of the feet. Certain complex acts that were initiated before the alteration of consciousness such as walking, chewing food, turning the pages of a book or even driving may continue. However if asked specific question or given a command, the patient is obviously out of contact. There may be no response at all or the patient may look towards the examiner in a perplexed way or utter a few stereotyped words. The patient is thus confused and in an irritable state, may resist or strike out at the examiner.

The violence and aggression that are said to characterize patients with temporal lobe epilepsy. Unprovoked assaults or outbursts of intense rage or blind fury are unusual. Rarely laughter or roaming may be the most striking feature of an automatism **(Gelastic epilepsy and Epilepsia procusiva respectively)**; or simply wander aimlessly either as an ictal or postictal phenomenon (Poriomania). Dystonic posturing of the arm and leg contralateral to the seizure focus is found to be a frequent accompaniment. After the attack, the patient usually has no memory about what was said or done. Any type of CPS may proceed to tonic spasm or other forms of secondary generalized seizures.

Seizures of temporal lobe origin can be at times confused with a number of psychiatric conditions like hypomania or schizophrenia. Some patients will lapse into paranoid, delusional or amnesic psychosis for a few days or weeks. EEG during this period may show no seizure activity in the Amygdala and other deep temporal lobe structures. Psychiatrists make the diagnosis of temporal lobe epilepsy rather fairly. Helpful in diagnosis is the known occurrence of seizures, amnesia for some of the events of the psychosis and seizure discharges in EEG. MRI will disclose a temporal lobe lesion CPS in about a third of cases. Postictal posturing and paresis of an arm or aphasia is helpful in detecting the side of lesion.

The third or last phase in CPS is the phase of partial responsiveness with reactive automatism. This phase can last for 5 minutes, the patient being confused usually for two to three minutes with an individual patient tending to have seizures of the same duration on each occasion.

C. PARTIAL STATUS EPILEPTICUS:

Inhibitory factors particularly of the cortex and the thalamus usually terminate focally originating motor seizures after one to two minutes. In other circumstances, excitation overwhelms inhibition, allowing extensive propagation and secondary generalization. When neither side wins this excitatory-inhibitory struggle, a prolonged re-iterative partial motor seizure results. Several varieties have been described.

The first variety described by Kojevnikov and termed Epilepsia partialis continua, consists of repeated jacksonian seizures characterized by a motor march. The Second variety consists of persistent, stereotyped, focal or regional, periodic or quasi – periodic myoclonus without march. At the other extreme are repetitive attacks or Jacksonian march.

Epilepsia partialis continua also may be associated with the chronic focal encephalitis of Rasmeussen

D. COMPLEX PARTIAL STATUS; In this variety, continuous altered behaviour has been observed. Complex partial status epilepticus may consists

of alternating phases of total unresponsiveness and speech arrest with stereotyped automatisms and partial responsiveness, with partial speech arrest and quasi-purposive automatisms. At times, a complex partial status may result in wandering, a condition termed **"fugue state".** Seizures may be confused with psychiatric disease or metabolic or other encephalopathies with delirium, i.e. Delirum tremens. A sudden alteration of behaviour, particularly in patients with a previous history of epilepsy should raise this possibility. The usual site of origin is mesial temporal and the limbic structures or the frontal areas.

INVESTIGATIONS IN PARTIAL EPILEPSY:

Electroencephalography:

Next thing which helps us to localize in the evaluation of seizures is Electroencephalography, EEG is complementary to CT & MRI.

Focal spikes, focal polymorphic delta waves, frequency difference between 2 hemisphere > 1 Hz, Focal slow waves (FIRDA), occipital sharp waves, phase reversal, onset of spike wave in EEG have localizing value.

Polymorphic delta waves	-	Superficial space occupying lesion
Rhythmical slow waves	-	Deep SOL
FIRDA	-	Midline tumours
Frontal sharp waves	-	Jacksonian or versive

Parietal sharp waves	-	Versive or sensory
Anterior Temporal sharpwave	-	Complex partial
Midline sharp waves	-	Simple partial
PLEDS	-	Epilepsia partialis continua

If EEG of a person shows anterior temporal spike or sharp wave it is strongly associated with the occurrence of clinical focal onset of seizure. When this pattern is seen on EEG likely hood of the person developing seizure is 98%. Converse is not true – proved in study by Joseph F. Hylihan M.D. mentioned in his article focal EEG wave form abnormalities**.(9)**

Focal polymorphic delta waves were often (68%) associates with

focal structural lesion in CT brain. Stroke being the most frequent etiology**.(10)**

In patients with normal CT brain in a study of 100 cases of focal delta activity convulsion itself was the most common cause with the exception of Mesial Frontal lobe epilepsy. Ictal EEG recordings are very useful in the

localization/ lateralization of focal seizures falsely (28%).**(11)** Some studies revealed that many patients with generalized tonic clonic seizure have focal features. Structural, functional imaging studies as well as histopathological studies have show presence of focal brain abnormality in patients with generalized epilepsy. **(12)**

In many situations EEG in partial seizure may be totally Normal. Hence if it is abnormal above mentioned wave forms may help us to localize. On the contrary Normal EEG doesn't rule out focal onset. Many a time the surface EEG is normal in partial seizure, because a critical area of 6 cm^2 has to be involved for the scalp electrode to pick up focal abnormality in the EEG.

When a patient has got only partial motor seizure the chance of EEG being abnormal is only 33%, if he has pure simple partial seizure with only sensory, the chance of EEG being abnormal is only 15% **(13)**

EEG as a tool for prognosis assessment has been studied in some papers published in International Journals, both with drugs and epilepsy surgery cases. Focal spikes and focal slow waves in every area is much frequent in patients with uncontrolled seizures than in patients who has very good control with antiepileptic drugs. People with frontal spikes also had poorly controlled seizures. This is shown in a work done by Jhon Hughes by reviewing 804 EEGs **(14)** .Patients with MRI and EEG findings which are concordant are more likely to be seizure free when compared to those who has a discordant when compared to those who has a discordant relationship. About 72% of patients were seizure free after surgery when they have concordant finding compares to 41% those who had discordant findings**.(15)**

When the EEG of a patient with partial seizure is abnormal the chance of getting an abnormal CT brain is about 57.8% **(16).** Limitations of EEG with evaluation of seizures;

1. Standard scalp electrodes record from as little as 1/3 of cortex
2. Deep cortex are after too distant for scalp electrodes.
3. Scalp and skull serve as special averages further hindering EEG interpretation.

Wicket rhythm may be mistaken for epileptiform changes in temporal lobe.**(17)**

Magneto encephalogram and EEG complement each other for the detection of interictalepileptform discharges. EEG offers the advantage of long term recording significantly increasing its diagnostic yield which is not feasible with MEG. MEG is more sensitive for the detection of Neocortical spike sources and can clarify the spatial relationship between the irritative zone and structural lesion.

In the evaluation of focal seizure one interesting feature in EEG is mirrorfocus. Mirror focus does not alter the prognosis after the epileptic surgery.

NEURO IMAGING:

Next investigation which comes to our help is Neuroimaging. Both CT brain and MRI brain help us to identify the lesions producing epilepsy.

CT SCAN

There were many studies with CT scan brain with regard to epilepsy both Nationally and internationally.

In one study by Hussein et al published in the year 2004, CT brain was abnormal in 68% of their study population ie., patients with partial seizure. **(18).**

In their study 75/102 positive CT lesion causes showed single ring enhancing lesion, Parietal lobe being the commonest site for SCECTL in their study. Partial seizures with or without secondary generalization being the commonest seizure type in patients with single contrast enhancing granuloma. This is shown by Chopra et al 1992. **(19)**

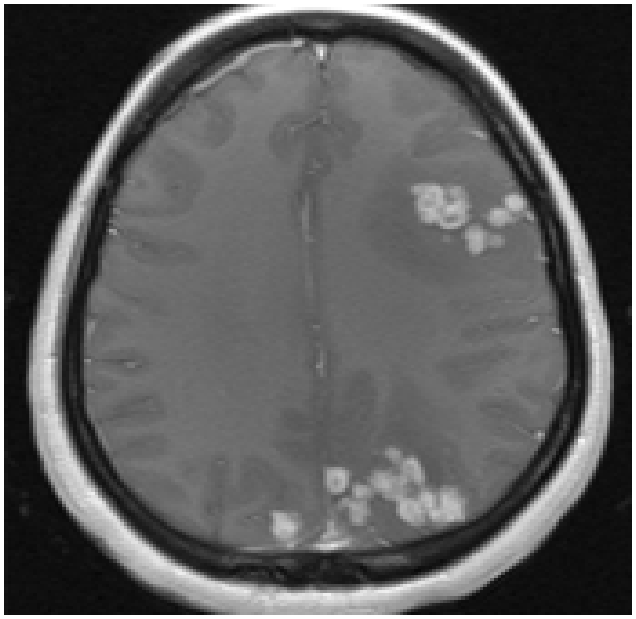
Vedhantham Rajasekar, Chandy et al has proposed following CT criteria to diagnose Neuro cysticercosis.

1.	Solitary	2.	Enhancement with contrast
3.	Less than 20 mm	4.	Perilesional edema may or

TUBERCULOMA

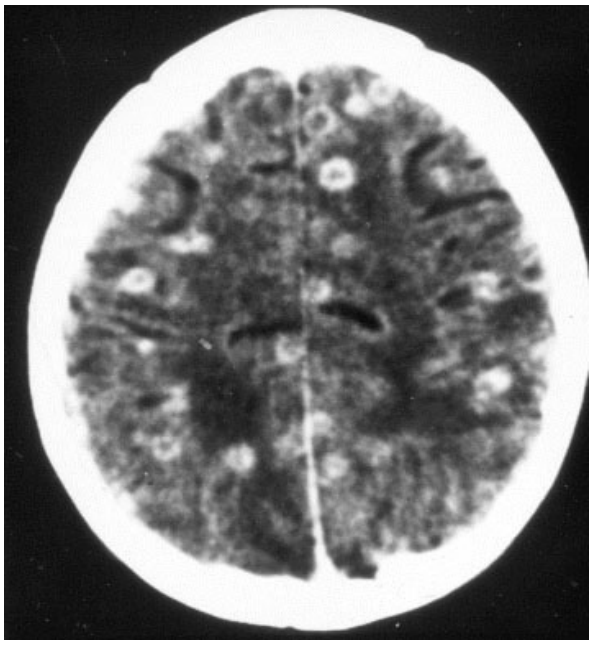
Parenchymal tuberculosis. (Contrast-enhanced CT scan shows multiple bilateral ring-enhancing lesions (tuberculomas) in the frontal and parietal lobes)

Parenchymal tuberculosis. Axial contrast-enhanced T1-weighted MR image demonstrates multiple enhancing caseating and non-caseating tuberculomas, predominantly within the left frontal and parietal lobes.

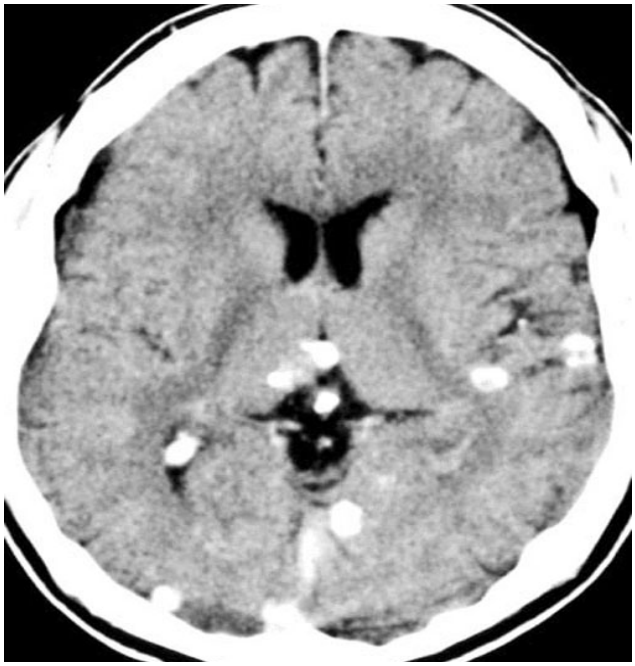


NEUROCYSTICERCOSIS

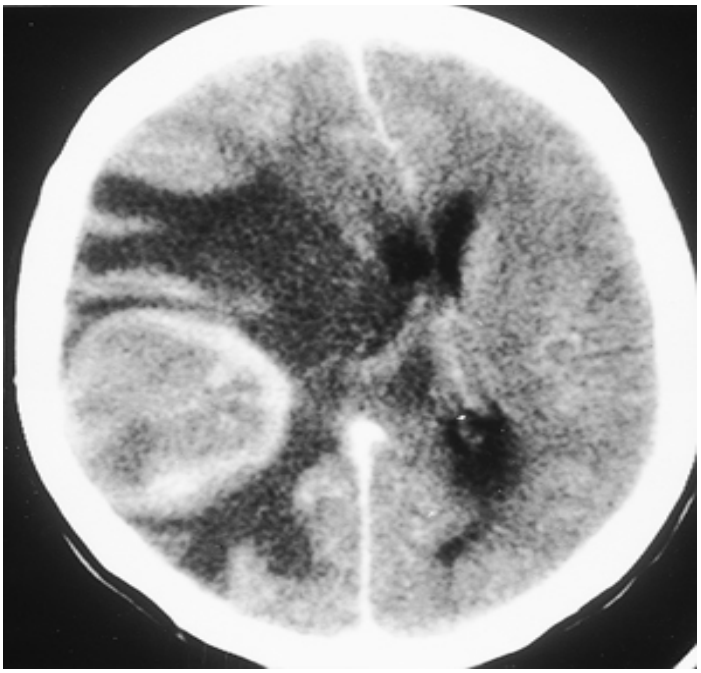
Enhanced CT scans of the brain in a patient with neurocysticercosis show multiple ring-enhancing lesions with perifocal edema.



Nonenhanced CT scan of the brain demonstrates the multiple calcified lesions of inactive parenchymal neurocysticercosis.



MENINGIOMA



POST STROKE SEIZURE-

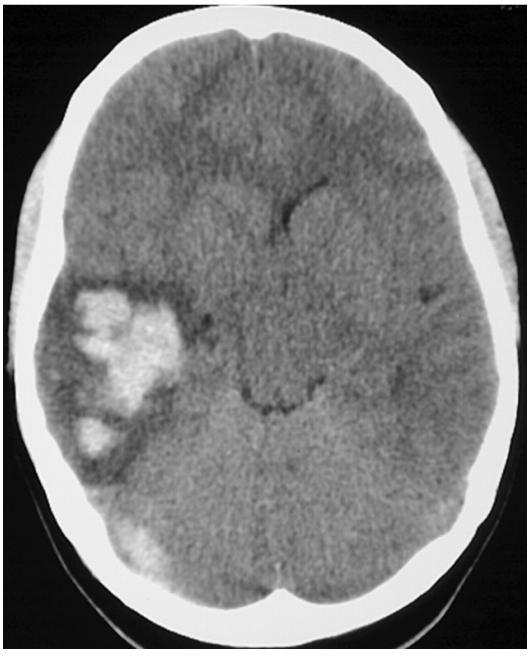
ICH WITH MIDLINE SHIFT

INTRACEREBRAL INFARCT

CORTICAL VENOUS THROMBOSIS

CT venogram showing a filling defect in the sagittal sinus (black arrow)- "Empty Delta sign"

Follow up CT scan of the head demonstrated hemorrhagic conversion of the infarct



May not be present but if present it must be without midline shift.

With thin slice contrast CT scan brain sensitivity of detecting such lesions is about 98%. Hence he concluded contrast CT is a reliable and cost effective modality to diagnose one of the commonest cause of seizure in this part of world. He even, quotes that "use of CSF for Immunological test to diagnose NCC is not recommended when clinical and CT features are generally straight forward **(20).** Misra et al in the year 1994, in their study of CT observation in partial seizures ,they found CT was abnormal in 79.3% of patients with partial seizures done at BHU – Varanasi, Commonest lesion being focal disc or ring enhancing lesion (63.3%) followed by calcification (11.8%) **(21).** Zee et al in the year 1980 were the first to report solitary contrast enhancing granuloma on CT scan brain. SCG are classified as disc enhancing, ring enhancing, and doughnut lesions.

Disc lesion - Uniform enhancement

Ring lesion - Peripheral enhancement with central hypodensity

Doughnut lesion - Peripheral enhancement occupies much greater area leaving a small central hypodense area.

Dot inside the lesion represents the scolex. Cases with scolex inside the lesion respondents better to albendazole therapy.

All these lesions if solitary are known as **Type A** lesions.

If there is a combination of disc and ring or 2 discs or 2 rings in a single CT brain it is called as **Type B** lesion with regard to NCC.

MRI AND OTHER IMAGINGS

MRI, PET, SPECT are also used in the field of epileptology, particularly with the introduction of epilepsy surgery. Role of MRI is very useful in identifying lesions which could be easily missed in CT evaluation like Neuronal Migrational disorders and vascular malformations. Sensitivity of MRI approaches 100% in tumour, vascular malformations, infarcts, granuloma.

But not only conventional MRI but also some newer techniques like quantitative T2 relaxometry, diffusion Tensor imaging, double inversion recovery, fast flair T2 image, Magnetization transfer technique, MR spectroscopy has to be used in patients particularly having refractory partial seizures before subjecting to epilepsy surgery, because conventional MRI may fail to identify a cerebral lesion in 20% patients with refractory partial seizure. Quantitative evaluation of T2 images is more sensitive and objective than visual assessment for identification of sublet pathologies **(22,27).** Role of MRI is valuable in the diagnosis of mesial temporal lobe sclerosis, Focal cortical dysplasia. 90% of patients non lesional temporal lobe epilepsy,localization of ictal onset zone is in the amygdale or hippocampus **(23)**

Patients with MRI identifiable structure lesion may be triaged to epilepsy surgery early in the course of treatment if it is clear that the initial response to anti epileptic drugs are disappointing.

PET studies utilizing FDG is mainly used in Pre surgical evaluation of patients with refractory partial seizures. It shows diffuse/ regional hypometabolism in 90% interictal recording and some regional hypoperfusion. Periictal PET shows diffuse/ regional hyper metabolism. Presence of temporal hypometabolism and absence of extra temporal cortical hypometabolism predicts best outcome in temporal lobe epilepsy **(24).** Pseudo PLEDS on scalp EEG can be associated with focal hypermetabolism even in the absence of overt seizure. This suggests in some who are experiencing clinical seizure manifestation of PLEDS may be an ictal rather than a interictal EEG pattern **(25).**

f MRI has also been utilized in Pre surgical evaluation to identify eloquent areas. ^{11}**C** Flumazanil PET is a newer technique which gives higher yield than the Advanced MRI technique in picking up the epileptogenic areas. Carbon 11 labeled flumazenil is a marker for the functional integrity of the GABAnergic inhibitory system. Loss of GABAnegic blinding by ^{11} **C** Flumazanil PET shows the areas of epileptogenic zone. But its high yield is not directly transferable in assessing the surgical results. There is study by Mathias J Koepp et al. Among 102 patients, with MRI negative partial seizures ^{11} **C**

Flumazanil PET abnormality but these findings were of use for surgery only in 25% of patients (26) . Because these picks up some white mater abnormalities like micro dysgenesis (increased density of heterotopic white matter neurons) which are not easily respectable by surgery. Moreover epileptogenic zone is the area of cortex necessary for seizure generation, which according to Rosenow no technique helps us to measure it directly and accurately (27). Area of seizure onset might be same or smaller and sitting inside the epileptogenic zone.

MANAGEMENT:

Patients with partial seizure who doesn't have an identifiable lesion have best prognosis.

Satisfactory results (an acceptable number of seizures or none, acceptable side effects) are obtained in approximately 70% of patients with a single drug, either the initial choice or an alternative. 30% of patients have inadequate control despite trials of several drugs used alone. When a second drug is added, another 10% are satisfactorily controlled. With a third drug, another 5% are satisfactorily controlled. Approximately 15% of patients are not controlled even after trails of three or more drugs. Such patients are considered medically refractory.

Besides patients without structural lesion, patients with SCG also respond well to Medical Management. Focal seizures in younger age groups have a better control over older age group.

Usually patients with SCG needs short term AED only for about 6 months. (28). But people with calcified granuloma need prolonged AED. In such cases therapy has to be individualized. Role of albendazole in SCG has been much debated. CT brain with ring enhancing lesions with dot inside respond better to albendazole. In other cases spontaneous revolution is possible, needs only AED and repeat CT brain after 3-6 months as per Rajashekar et al studies at CMC Vellore. Gliosis around focal cerebral calcification as seen in TI Magnetisation Transfer MRI is a prediction of poor seizure control.

Patients with acute symptomatic partial seizure due to metabolic insults like hyperosmolar nonketotic coma, hypocalcemia responds very well to the correction of underlying abnormality. They do not need long term AED therapy.

MEDICAL TREATMENT

The following anti epileptic drugs are commonly used in the treatment of partial seizures. They are carbamazepine, oxcarbazepine, Gabapentin, phenytoin, Sodium valproate, Topiramate, Lamotrigine, Tiagabine, Leviteracetam. Carbamazepine or phenytoin is currently the initial drug of

choice for the treatment of partial seizures including those with secondary generalization. Carbamazepine is preferred because of its pharmaco kinetics and toxicity profile (29). Valproic acid is an effective alternative for some patients when particularly the seizure secondarily generalize.

Gabapentin as an add on therapy in partial seizure in patient who are not responding to monotherapy is been well studied. In India, Prof. Dhanraj has established its role as an add on therapy in partial seizure in his paper published in the year 1998. **(30)** Gabapentin is unique in that it does not have any significant drug interaction. Lamotrigine is almost effective in all sub types of partial seizure. **(31)** Lamotrigine appears to have an over all efficacy profile similar to the more standard drugs and is now being used as mono therapy and also can be used as an add on therapy. When used as an add on therapy does should be started as a lowest possible and slowly titrated up.

Antiepileptic Drugs of Choice:

Seizure type	Drugs of first Choice	Drugs of second choice	Alternative drugs
Partial (simple, Complex, secondarily generalized tonic-clonic)	Carbamazepine Phenytoin	Gabapentin Lamotrigine Topiramate Valproic acid	Phenobarbital Primidone Tiagabine

PHARMACOKINETICS

	Responder Rate	No Serious Toxicity	No Nuisance toxicity	No drug inter action	Administrations
Gabapentin	30-40%	+	±	+	t.i.d
Lamotrigine	30-40%	-	±	±	b.i.d
Phenobarbital	?	+	-	-	q.d
Primidone	?	+	-	-	t.i.d
Tiagabine	20-30%	-	-	-	b.i.d or t.i.d
Topiramate	40-50%	-	-	+	b.i.d
Valproic acid	30-40%	-	-	-	b.i.d. or t.i.d

EPILEPTIC SURGERY:

The following surgeries are done for patients with medically refractory seizures.

1. Temporal lobectomy
2. Non temporal resections
3. Corpus callosotomy
4. Hemispherectomy

5. Subpial resections

6. Lesionectomy

In a statistics from a tertiary referral centre in India showed that about 74% of patients with intractable seizure referred to surgery were suffering from partial seizures. **(32)**

Clear identification and complete resection of epileptogenic focus will result in good outcome. Almost 90% of people will become seizure free in Mesial temporal lobe epilepsy **(33).** In children operated for epilepsy with tumours, after the resection of tumour almost 90% of them became seizure free **(34).** In the study at CMC vellore by Danial et al over 40 years period showed, total or near total control in 35% of patients and worth while outcome in another 25% **(35)** . In western centres they have noted 70-80% seizures control after epileptic surgery.

Post operatively patient generally need to remain on antiepileptic drug therapy but the marked reduction of seizures following surgery can have a very beneficial effect on their quality of life.

PROGNOSIS OF PARTIAL SEIZURE AND LOCALIZATION RELATED/SYMPTOMATIC EPILEPSIES

A. First Unprovoked Seizure:

The risk of seizure recurrence by 36 months is 25% in persons with no risk factors after having a first unproved seizure. In persons with risk factors,

the risk of seizure recurrence usually is much greater. Risk factors include evidence of prior neurologic insult (determined by history, neurology examination, imaging studies), abnormal EEG, and multiple seizure or status epilepticus as initial event. Treatment after first partial seizures remains controversial because of the uncertainty regarding the risk of another seizure and the side effects of antiepileptic medication. However, randomized clinical trials do indicate that antiepileptic drugs reduce risk of seizure recurrence.

B. After two or More Unprovoked Seizure:

Persons with two or more unprovoked seizures almost always are treated. The two Veterans Administration Cooperative studies indicate that 35% to 60% of adult patients with partial seizures will have complete seizure control after 1 year with carbamazepine or phenytoin monotherapy as the initial and only treatment.

Satisfactory results (an acceptable number of seizure or none, acceptable side effects) are obtained in approximately 70% of patients with a single drug, either the initial choice or an alternative. Thirty percent of patients have inadequate control despite trials of several drugs used alone. When a second drug is added, another 10% are satisfactorily controlled. With a third drug, another 5% are satisfactorily controlled. Approximately 15% of patients are not controlled even after trails of three or more drugs. Such patients are considered medically refractory.

Risk factors for poor control of partial seizures include.

Abnormal EEG,

Evidence of a structural brain lesion,

Number and duration of seizures before diagnosis and before control with medication,

Neurologic deficit from birth, and

Secondarily generalized tonic-clonic seizures.

C)mortality:

1. General:

Available studies are not optimal but generally report increased mortality in patients with symptomatic epilepsies. This mortality is caused, at least in part, by the underlying symptomatic disease (congenital malformations, tumors, cerebrovascular disease) and its complications. Studies regarding increased rate of suicide are conflicting.

2. Sudden Unexplained Death:

The risk of sudden unexplained death is between 1 in 500 and 1 in 1,100 person years for all persons with epilepsy, and 1 in 200 person – years for patients with refractory seizures. The risk is greater for persons between the ages of 15 to 45 years with poorly controlled tonic-clonic seizures (usually

secondarily generalized). Structural lesions and severe or frequent seizures appear to be risk factors. Available evidence suggests that most sudden deaths are temporally related to seizures and often occur in sleep. Postulated mechanisms include cardiac arrhythmias, pulmonary edema, and suffocation.

D. Neuropsychologic Function:

Animal studies suggest that repeated partial seizures may result in neuronal damage. Human studies are difficult to evaluate because of the confounding effects of the original structural lesion, antiepileptic drug effects, and impaired social adjustment. The effects of repeated partial seizures on neuropsychologic function remain unknown

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was carried out in the Department of Medicine and Department of Neurology, Coimbatore Medical College Hospital, Coimbatore

INCLUSION CRITERIA

Total of 50 patients who were admitted with history of focal seizure or attending Neurology OPD were included in this study.

EXCLUSION CRITERIA

Patients with a history of recent head injury and those who were admitted in the surgical ward of the hospital were excluded.

STUDY PERIOD

From April 2009 to September 2010.

TYPE OF STUDY

Descriptive Study

Detailed history was elicited (Both from the patient and his/her intelligent attender) and clinical examination was carried out to ensure the organic nature of epilepsy. A proforma formulated by the Post graduate and

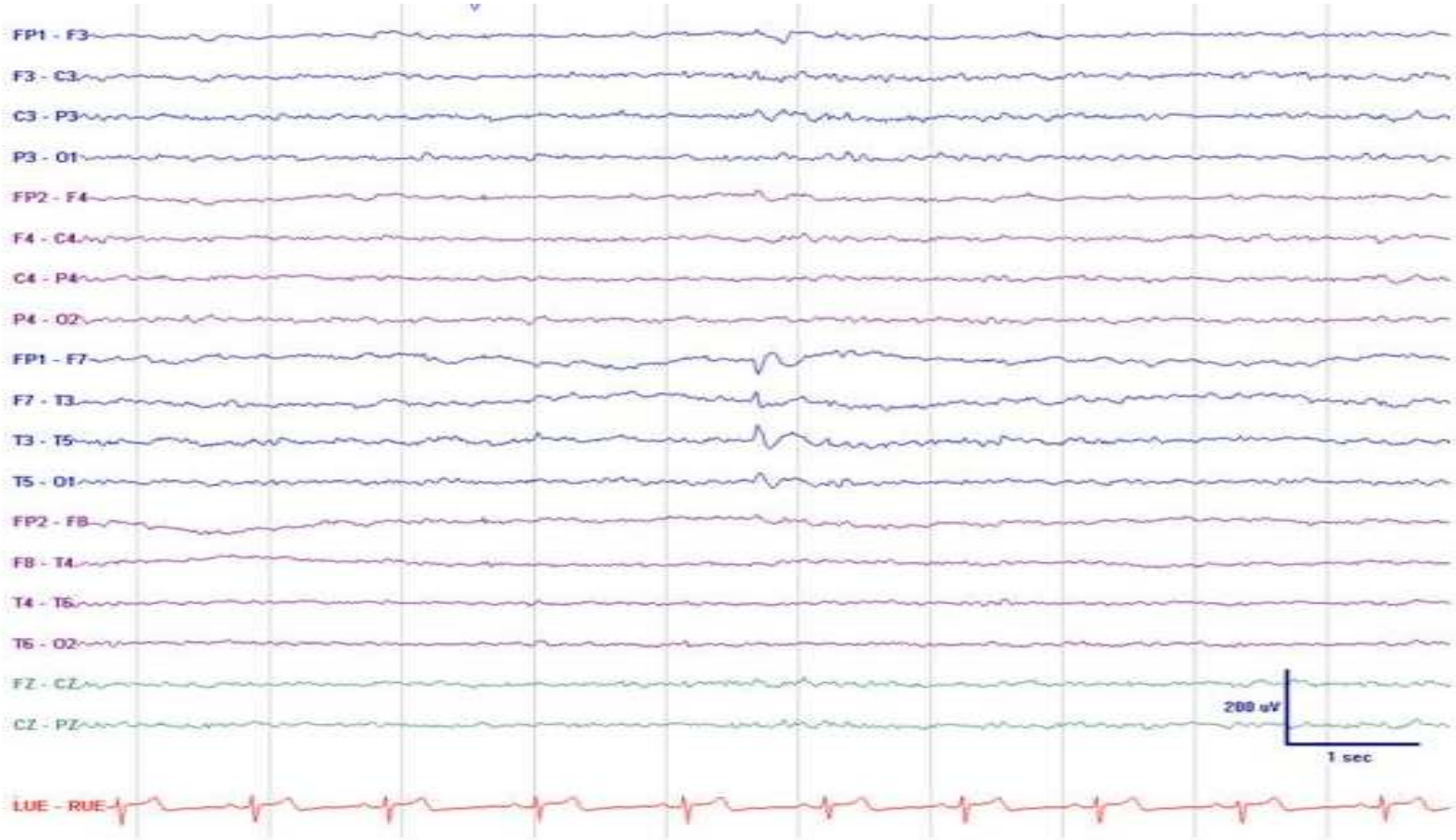
accepted by the Professor of Medicine was used to collect the data –Proforma enclosed.

All the EEGs were critically analyzed for the presence of focal, localized or generalized changes by montage wise analysis.Individual abnormalities were recorded in the proforma .

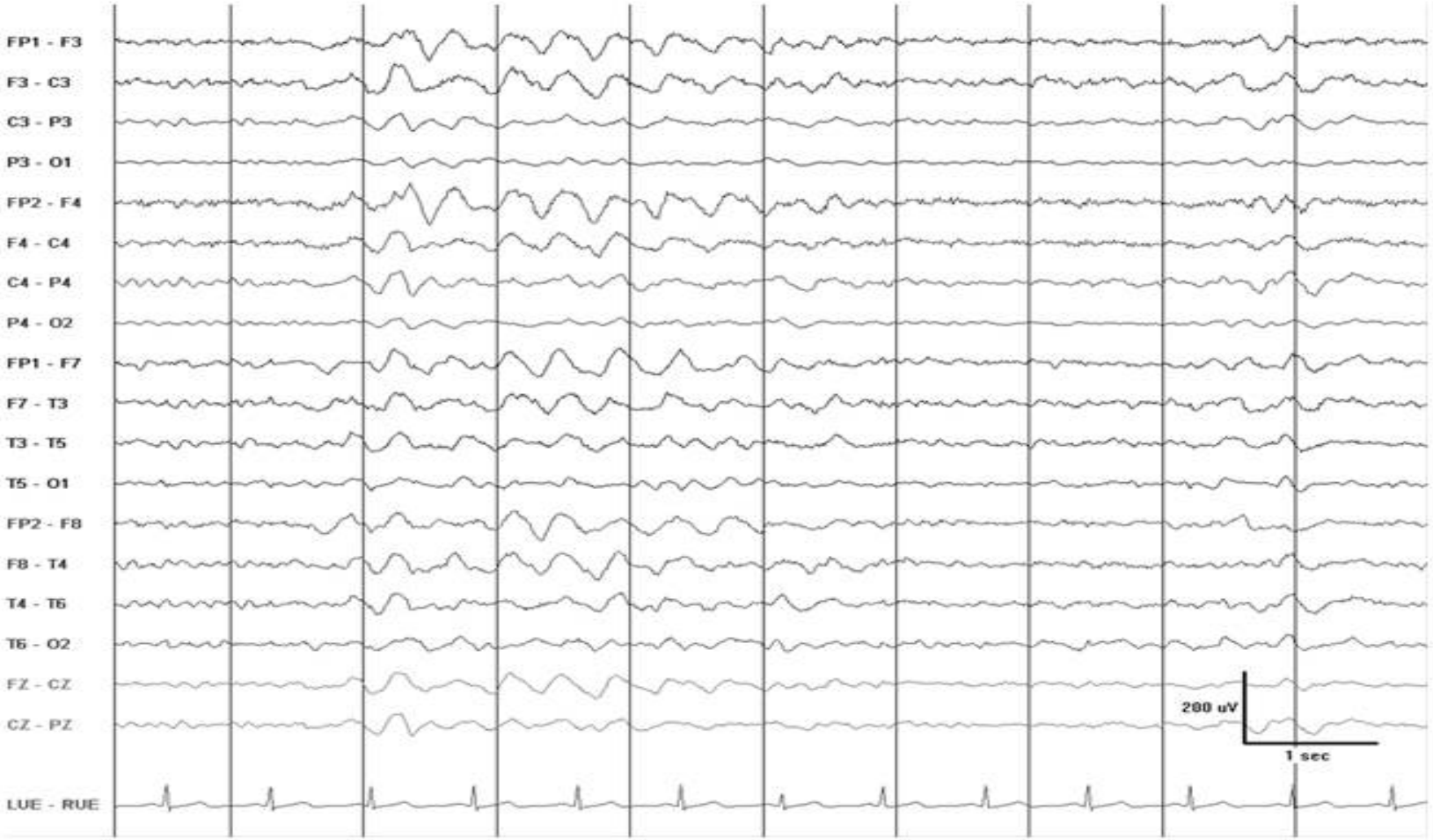
CT scan brain plain and contrast axial section with routine slice thickness was performed in all cases. Radiologist opinion obtained. Abnormalities noted.

Finally the data was analyzed combining the clinical EEG and CT scan brain findings and conclusion arrived.

Right posterior temporal spike. This EEG is from a 17-year-old boy with a history of complex partial seizures. The spike phase reverses at T6. The field of the discharge extends into the parietal and occipital regions.



Left anterior temporal spike. This EEG was recorded in the drowsy state. The patient was a 54-year-old man with a history of monthly episodes of behavioral arrest and automatic behavior consistent with complex partial seizures arising from the temporal lobe.



Frontally predominant intermittent rhythmic delta activity (FIRDA) in a 70-year-old man with a metabolic encephalopathy.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

In this study total of 50 patients were examined , in them detailed history, clinical examination and Investigations were performed.

The incidence of focal seizures in the different age groups and the sexes were as follows:

AGE & SEX INCIDENCE

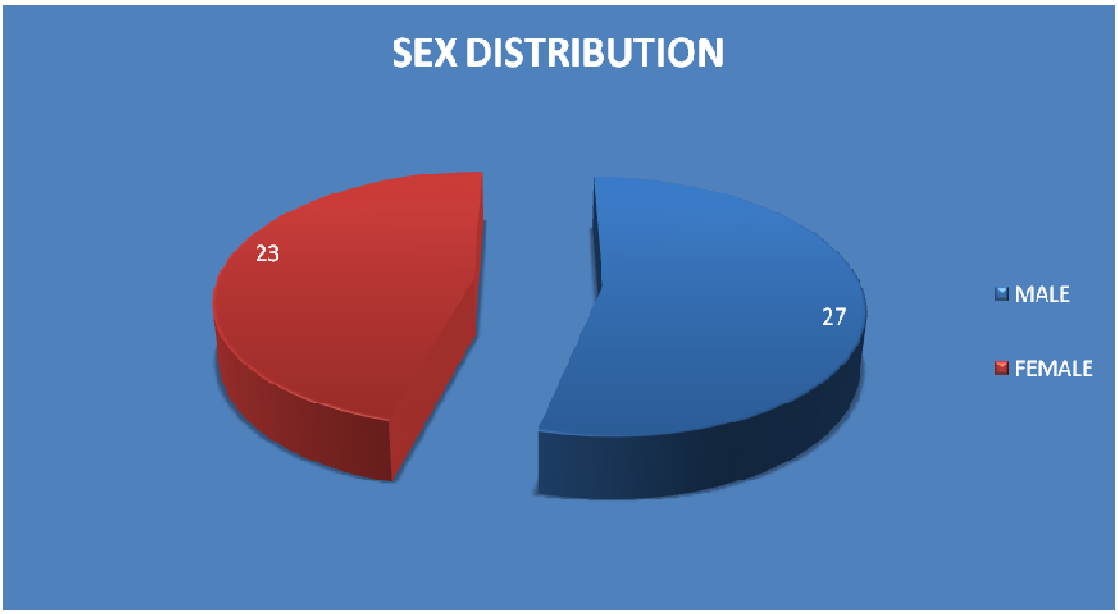
Table-1

Age Group	Male	Female	Total
0- 9years	3	2	5(10%)
10-19	8	5	13(26%)
20-29	3	6	9(18%)
30-39	2	5	7(14%)
40-49	1	3	4(8%)
50-59	3	1	4(8%)
>60	7	1	8(16%)
Total	27	23	50

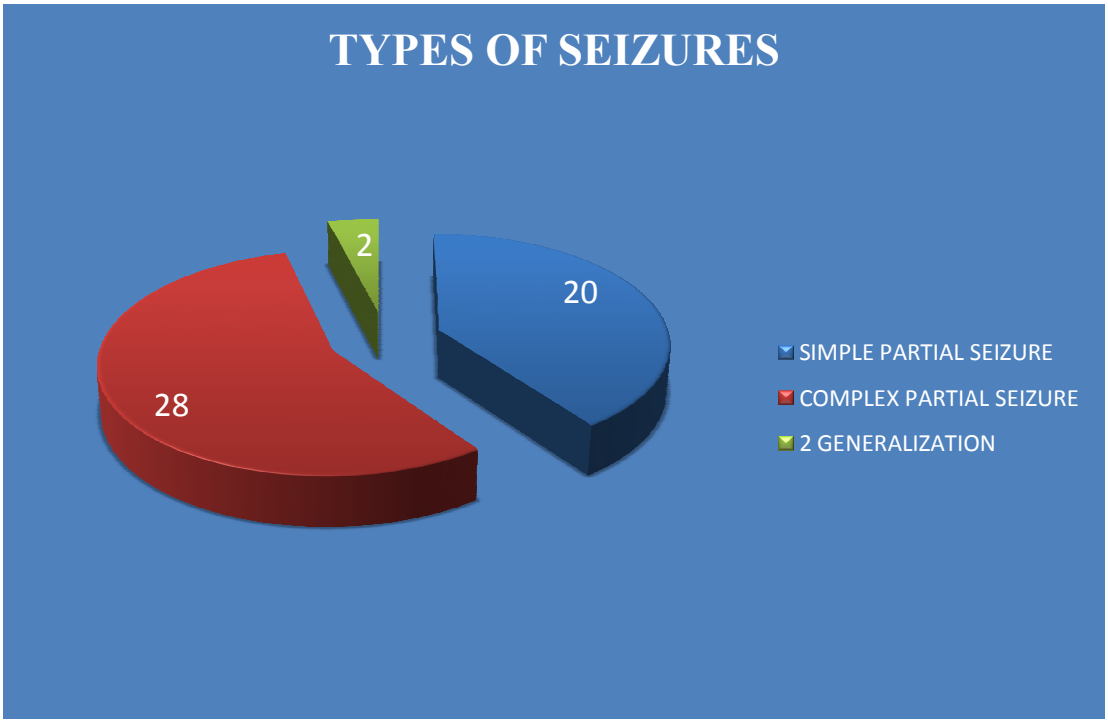
Among the total 50 cases, 20 patients had simple partial seizures, 28 patients had complex partial seizure and 2 patients had 2° Generalization.

Right focal seizure was noted in 29 patients and left focal seizure in 21 patients .

SEX DISTRIBUTION



TYPE OF SEIZURE



The number of attacks that each patient had was tabulated in relation to the age groups.

Table -2

AGE GROUP	NO OF EPISODES			
	1 episode	**2-5 episodes**	**6-10 episodes**	**>10 episodes**
0-9 years	2	3	-	-
10-19 years	3	7	2	1
20-29 years	1	5	1	2
30-39 years	1	2	2	2
40-49 years	-	3	-	1
50-59 years	-	3	-	1
> 60 years	3	5	-	-
Total	10	28	5	7

Of the fifty patients 42 (84%) had seizures while they were awake, 3 (6%) while they were asleep and 5 (10%) had both during sleep and awake period.

AURA

Of the fifty patients, only 8 (16 %) had "Aura", one patient had 3 forms of aura- sensory, visual and auditory. The distribution of"Aura" was as follows:

Table-3

AURA- INCIDENCE			
AGE GROUP	**Sensory**	**Visual**	**Auditory**
0-9 years	1	1	1
10-19 years	3	-	-
20-29 years	-	1	-
30-39 years	1	-	-
40-49 years	1	-	-
50-59 years	1		-
>60 years	-	-	-
Total	7	2	1

Only 6 patients had "**precipitating factors**"

These were as follows:

Fever : 2

Emotional Disturbance	:	1 (pseudo – seizure)
Chronic Alcoholism	:	1
Post-Partum dehydration	:	1
Anticonvulsant withdrawal	:	1

12 Patients had significant illness in the past. They were:

Diabetes mellitus Type I	:	1
Diabetes Mellitus type II	:	2
Hypertension	:	4
Tuberculosis	:	1
Ischemic heart disease	:	1
Sexually transmitted disease	:	1
Birth asphyxia	:	1

ABNORMAL LAB

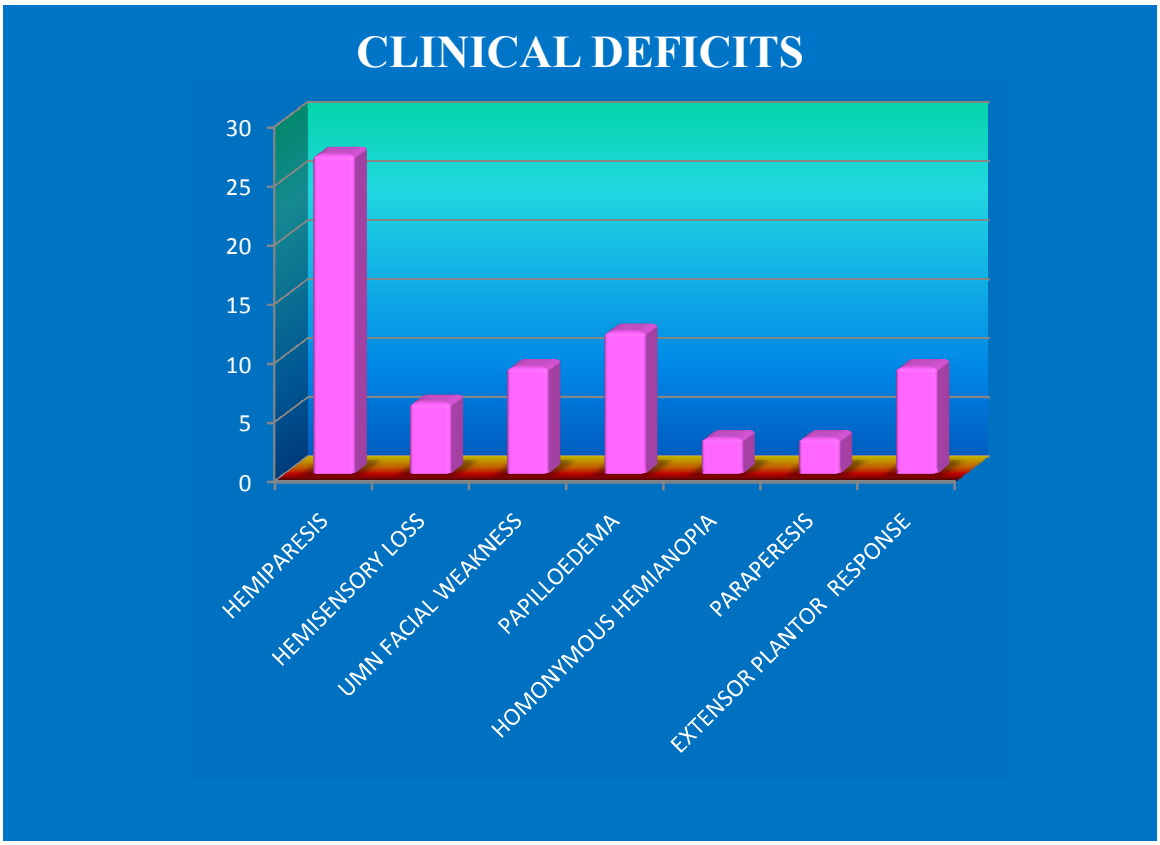
Hyperglycemia	:	2
Raised ESR	:	4
Hyponatraemia	:	1

CLINICAL SIGNS

After analyzing the symptoms of the patients, headache was the most frequent symptom and it was reported in 21 cases. And 23 patients out of the 50 cases had clinical signs of deficit. (46%). Among the clinical signs hemiparesis was seen in 9 patients (18%).2 patients had hemisensory deficit (4%). 3 had facial weakness of upper motor neuron type. 4 patients had papilloedema 8%).1 patient had homonymous hemianopia (2%). One patient had Paraparesis (2%). 3 had extensor plantar (6%)..

Table-4

S.No	Clinical Deficit	No.of patients	% of total
1	Hemiparesis	9	39.13%
2	Hemisensory impairment	2	8.69%
3	UMN facial weakness	3	13.04%
4	Papilloedema	4	17.39%
5	Homonymous hemianopia	1	4.34%
6	Paraperesis	1	4.34%
7	Extensor plantar response	3	13.04%
	Total	**23**	**100%**



In toto among the 50 patients who were examined - 23 Patients had deficit which amounts to 46%..

CT BRAIN

In this study, CT brain was abnormal in 37 patients (74%). 20 patients among these 37 patients had deficits on clinical examinations (54.05%). 17 patients with CT brain abnormality didn't show any deficit.

Among the CT brain lesion – Ring enhancing granuloma was the

most common lesion reported in 20 cases (54.05%). Among these, single contrast enhancing ring lesion was seen in 14 patients, disc enhancing lesion in 3, Double ring enhancing lesion in 1 and multiple ring enhancing lesion in 2. Two patients had shown scolex.

Infarct was seen in 6 cases (16.21%). Mass lesion was reported in 2 cases (5.4%). Calcification in 2 cases, Gliosis Arterio-venous malformation CVT, cerebral atrophy ,diffuse gyral enhancement each in one case.

Granulomas are most commonly reported in the parietal lobe. Among the 20 cases with granuloma, 17 patients had lesions in the parietal lobe. Distribution of granuloma was in the following order, Right parietal in 6 and left parietal in 11. Among the other 3 cases Temporal lobe, frontal lobe each one has one granuloma, other showed multiple lesions.

Among 20 contrast enhancing granulomas

Single ring enhancing lesion	-	14
Disc enhancing lesion	-	3
Double ring enhancing lesion	-	1
Multiple ring enhancing lesion	-	2

CT LESIONS

Table-5

S.No	CT Lesion	No.of patients	% of study
1	Contrast enhancing granuloma	20	54.05%
2	Infarct	6	16.21%
3	Mass	2	5.4%
4	Haemorrhage	2	5.4%
5	Calcification	2	5.4%
6	AVM	1	2.7%
7	CVT	1	2.7%
8	Cerebral Atrophy	1	2.7%
9	Gliosis	1	2.7%
10	Diffuse gyral enhancement	1	2.7%
		37	100%

Distribution of infarcts in the CT brain

Parietal - 4

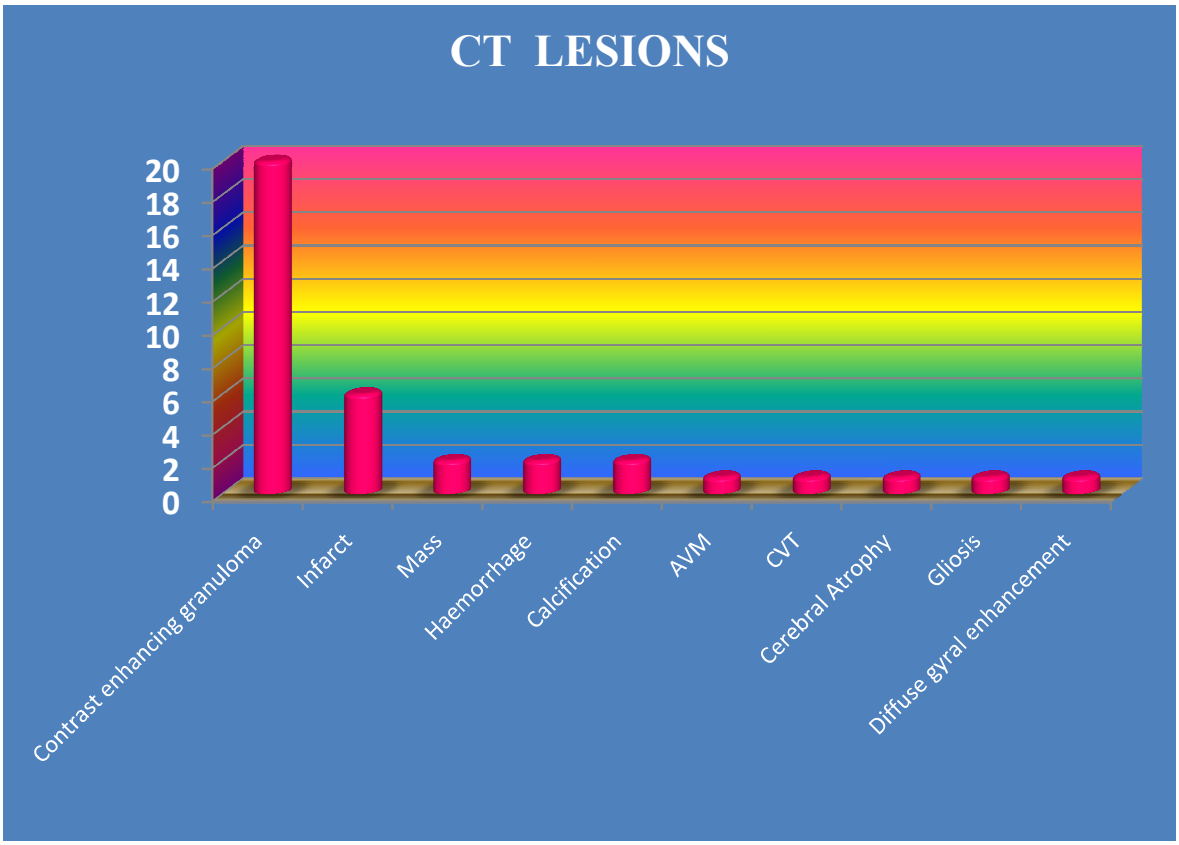
Temporal - 1

Occipital - 1

Distribution of mass lesion

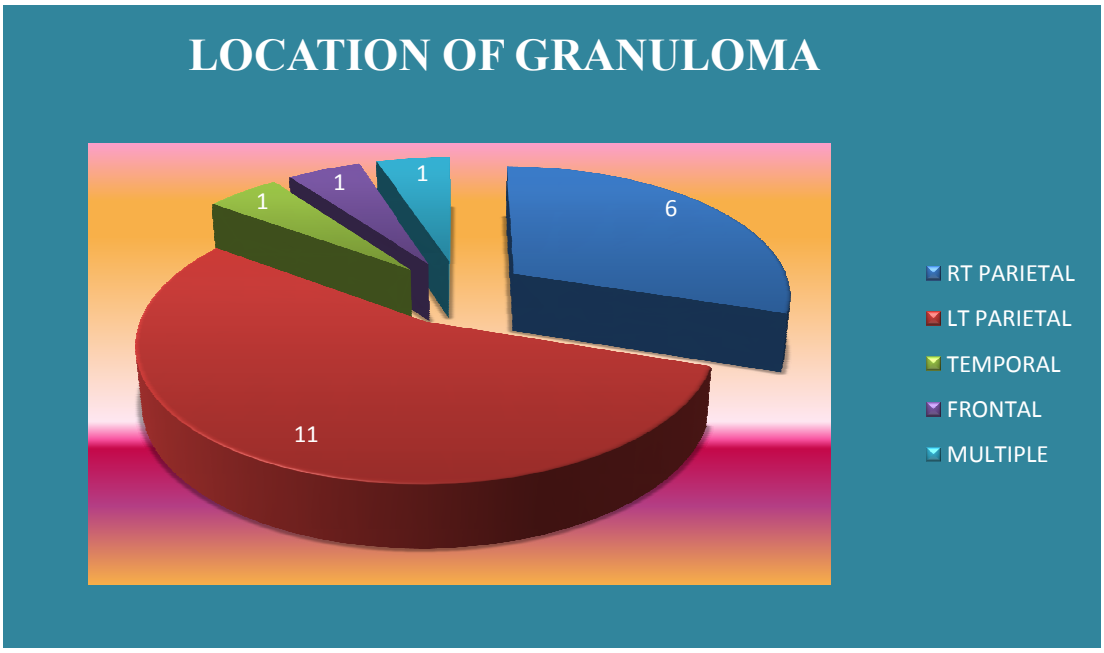
Suprasellar - 1

Parieto occipital - 1



Distribution of granulomas

Right parietal	- 6	Temporal	- 1
Left parietal	- 11	Frontal	- 1
		Multiple	- 1



Haemorrhage -2

Others

AVM - Parietal

2 Calcification - Parietal

1 Gliosis - Occipital

1 CVT

1 Cerebral Atrophy

1 Diffuse gyral Enhancement

Interestingly among the 37 cases with CT lesions almost in 24 cases lesions were seen in the parietal lobe.

EEG

EEG was abnormal in 27 cases (54%). Among these Generalized changes were present in 10 patients (20%). Lateralizing changes were present in 17 cases (34%)

Following were the EEG abnormalities noted

1. Phase reversal	-	7 cases
2. Bilateral spike, sharp waves	-	10 cases
3. Focal or unilateral sharp waves	-	5 cases
4. Focal slow waves	-	5 cases
Total cases with EEG abnormalities	-	27(54 %)
Lateralizing EEG Changes	-	17 cases
Bilateral changes	-	10 cases,

CLINICAL DEFICITS ,EEG & CT ABNORMALITIES-CORRELATION

CT CHANGES IN PATIENTS WITH CLINICAL DEFICITS

Among the patients with hemiparesis (9) CT brain was abnormalin 7 patients (77.8%).Among 2 patients with hemisensory deficit CT brain was abnormal in 1 patient (50%) .Among 3 patients with facial weakness of upper motor neuron type CT brain was abnormal in 3 (100%).

Among total of 4 patients with papilloedema CT brain was abnormal in all the 4 patients (100%).One patient with homonymous hemianopia CT brain was abnormal (100%). One patient with Paraparesis had CT abnormality showing a suprasellar mass lesion . CT was abnormal in all the three patients with extensor plantar (100%).

In total 50 patients who were examined, 23 Patients had deficit.CT brain was abnormal in 37 patients. 20 patients among these 37 patients had deficits on clinical examinations. 17 patients with CT brain abnormality didn't show any deficit.

STATISTICAL SIGNIFICANCE OF POSTICTAL NEUROLOGICAL DEFICITS IN PREDICTING CT BRAIN LESIONS

		CT Brain Lesion		
Clinical signs of Deficit		Present	Absent	Total
	Present	20	3	23
	Absent	17	10	27
	Total	37	13	50

X^2 = 2.57

P = <0.01

P →SIGNIFICANT

SENSITIVITY- 54 %

SPECIFICITY- 77 %

EEG CHANGES IN PATIENT WITH CT ABNORMARLITY

Among the patients with granuloma EEG was positive in 11 cases. So totally among 20 cases of ring enhancing granulomas as evidenced in CT brain, 11 patients had EEG abnormality (55%). Interestingly 10 patients

had shown lateralizing EEG abnormalities. (90.9%). Predominantly granulomas were seen in younger population.

Infarct was seen in 6 cases. Parietal lobe was the commonest site. 4/6 cases showed the infarct in the parietal lobe. EEG was abnormal in 3 cases (50%) of which 2 had lateralized EEG changes and one showed generalized changes.

Among the 2 cases with mass lesion, EEG was abnormal in 1 cases, both of them showed generalized changes.

Patient with CVT also showed generalized EEG changes. Patient with AVM, Cerebral atrophy showed lateralizing EEG changes. Patient with calcification, haemorrhage, gyral enhancement and gliosis didn't show any EEG Changes

Table-6

LESION IN CT	NO OF PATIENTS WITH CT LESION	NO OF PATIENTS SHOWING EEG CHANGES	PERCENTAGE
Granuloma	20	11	55%
Infarct	6	3	50%
Mass lesion	2	1	50%
Hemorrhage	2	0	0%
CVT	1	1	100%
Gyral enhancement	1	0	0%
Calcification	2	0	0%
AVM	1	1	100%
Gliosis	1	0	0%
Cerebral atrophy	1	1	100%
TOTAL	**37**	**18**	**48.64%**

So, totally among the 37 patients with CT abnormality 18 had shown abnormal EEG (48.64%) EEG was most commonly abnormal in patients with infective pathology than any other conditions. Lateralising EEG changes were also common in patients with infective Pathology

Table-7

ETIOLOGY

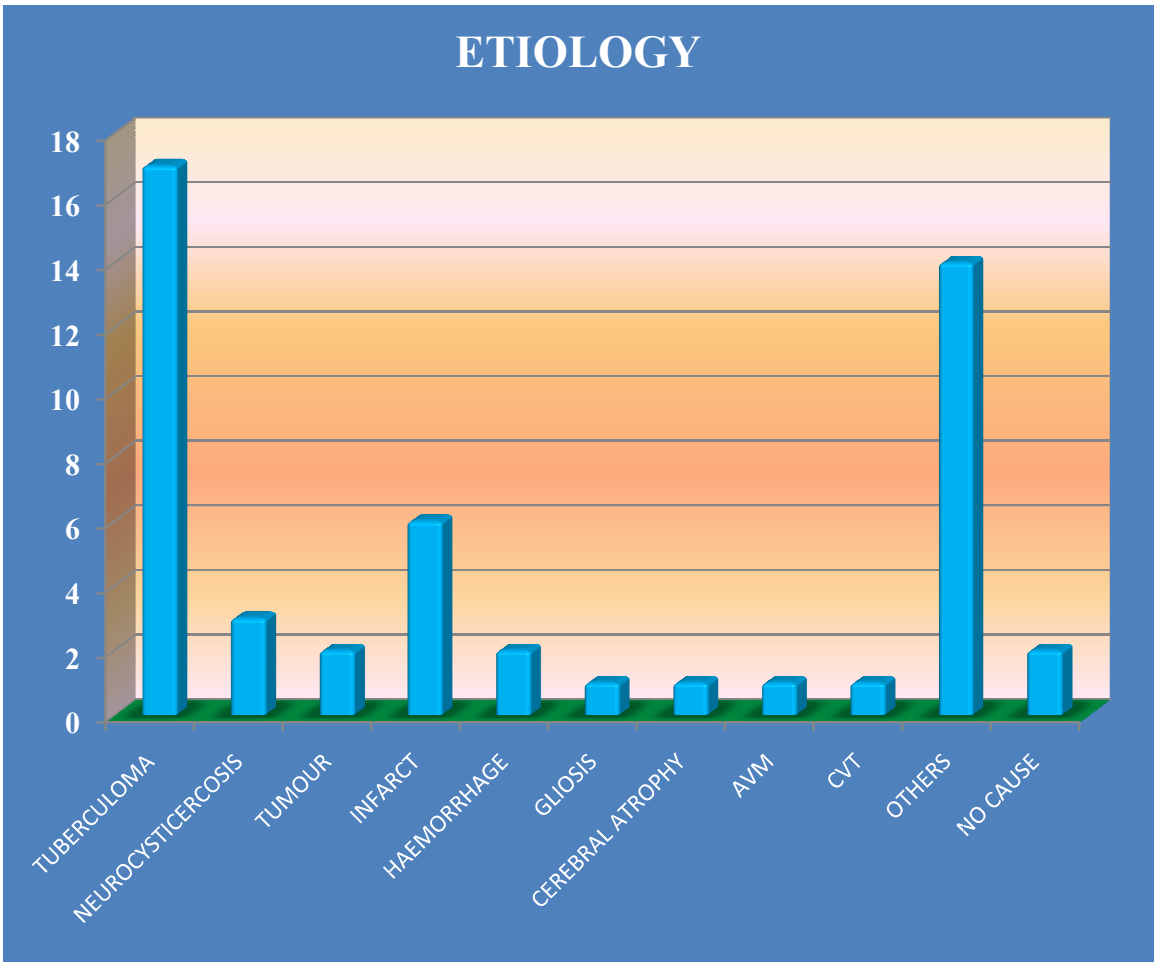
AGE GROUP	TUBERCULOMA	NEUROCYSTICERCOSIS	TUMOUR	INFARCT	HAEMORRHAGE	GLIOSIS	CEREBRAL ATROPHY	AVM	CVT	OTHERS	NO CAUSE
0-9 yrs	2										
10-19yrs	8									3	2
20-29yrs	3	2		1						2	
30-39yrs	4							1	1	2	
40-49yrs	-		1			1				2	
50-59yrs	-			2						1	
>60yrs	-		1	3	2		1			4	
	17	3	2	6	2	1	1	1	1	14	2

Others

Calcification - 2

Hyperglycaemia - 1

11 patients had abnormal EEG patterns


ETIOLOGY
18
16
14
12
10
8
6
4
2
0
TUBERCULOMA
NEUROCYSTICERCOSIS
TUMOUR
INFARCT
HAEMORRHAGE
GLIOSIS
CEREBRAL ATROPHY
AVM
CVT
OTHERS
NO CAUSE

DISCUSSION

DISCUSSION

A total of 50 cases were studied. Clinical history was taken as the tool for the diagnosis. Strictly conversion and other seizure mimics were excluded.

Computerized Tomography of brain was done in all 50 cases. Both plain and contrast CT brain axial section with normal window length / breadth with 10mm anterior and 5mm posterior cuts were taken. Radiologists opinion obtained in all cases. Vedhantham Rajasekar's criteria for NCC utilized.

Ring enhancing lesion is defined as peripheral thin rim of enhancement with central hypodensity and disc enhancing lesion is defined as uniform enhancing lesion in entirety. Mass lesions are not categorized into individual tumour.

Generalized EEG epileptiform activity and lateralizing EEG abnormalities were scrutinized. Among the lateralizing changes, phase reversal as defined as abnormal wave forms of opposite polarity in adjacent Bipolar montages. Focal spikes, sharp waves as defined by the duration < 70 m/sec, 70-200 m/sec respectively. Slow waves as defined by frequency less than 8 / sec were taken into consideration while interpreting the abnormality.

The sex incidence of partial seizures was almost equally distributed with a slight male preponderance. Complex partial seizure was the commonest entity. CPS : SPS = 1.4 : 1.Incidence of partial seizure is more common in younger population than in older. More frequent in young adults and children.When compared to generalized seizures, partial seizures are known to produce more clinical signs when evaluated postictally. In our study 23 cases out of the 50 (46%) had

positive clinical signs. Hemiparesis was the most frequent deficit noted.

Hemisensory, UMN facial weakness, homonymous hemianopia, papilloedema, extensor plantar response alone, paraparesis were also noted. After that study has been analysed whether people who has a deficit or sign on clinical examinations have a higher chance of harbouring a structural lesion in their brain when compared to those who don't have any deficit. And it was proved that those who have a deficit have more incidence of structural lesion than those who don't.

Among the patients with clinical signs or deficit postictally, 20/23 had structural lesion on CT brain. So, I conclude that patients with deficits are more likely to have abnormal CT brain. Headache was the most common symptom reported by patients. This history was given in 20 cases.

Among the 50 cases studied, EEG was abnormal in 27 cases (54%). 18 out of 27 patients with EEG abnormality had CT brain lesions. (48.64%). This is almost comparable to an Indian Study done in 2003 by Ramesh Bahti et al. In their study 57.9% of cases with EEG abnormality had abnormal CT brain. (15)

Generalized EEG changes were noticed in 10 cases and lateralizing EEG changes in 17 cases. 18 out of 27 patients with EEG abnormality had CT brain lesions. (48.64%). So in general patient with EEG changes in partial seizure are more likely to have structural lesion than those who don't. But in specific , patient with lateralizing changes have more chance of having structural lesion than generalized changes.

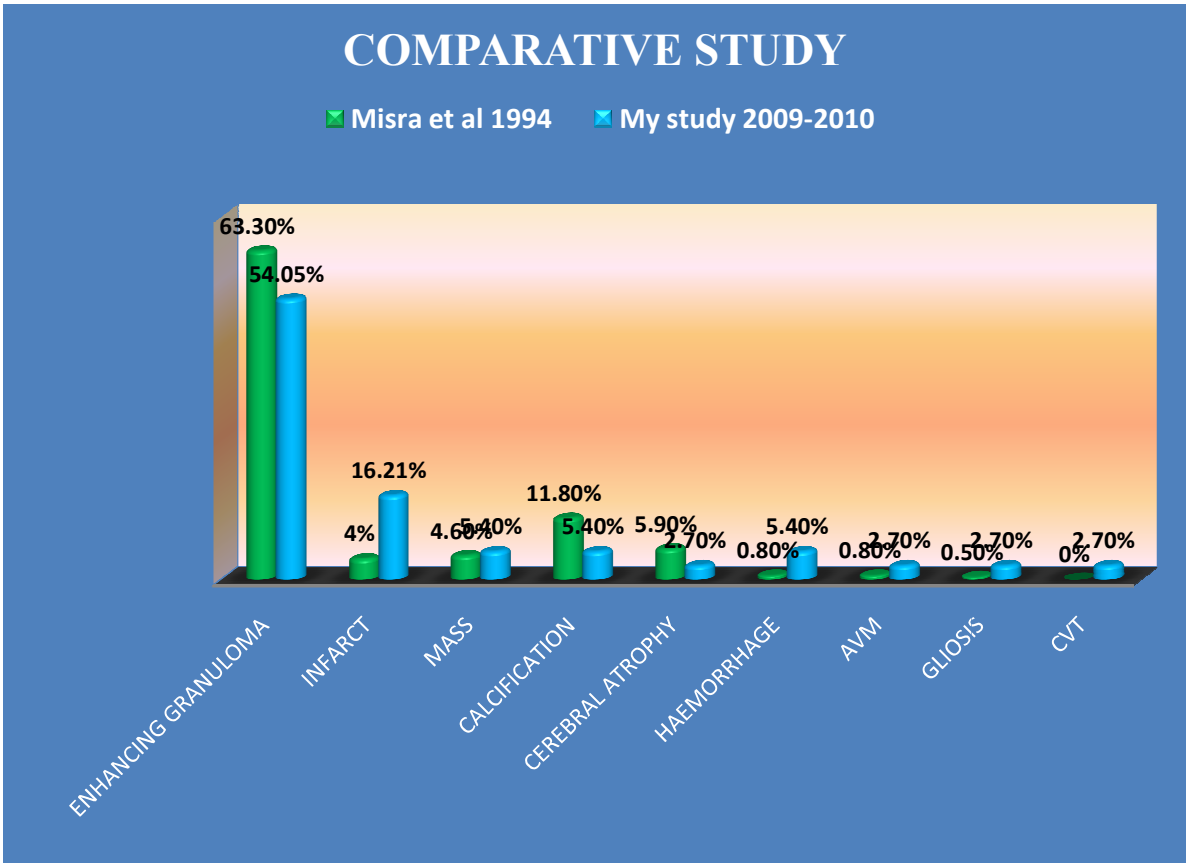
In the etiological aspect, contrast enhancing granuloma was the most frequent lesion. This Study had encountered contrast enhancing granuloma in 54 % of our study populations. In this study CT brain was abnormal in 74 %.It is comparatively lower than Misra et al study (1994) in which it was 79.3%(3)

Comparison of CT observation in Misra et al study in the year 1994 to this study. (3)

COMPARATIVE STUDY

Table-8

	Misra et al 1994(%)	*My study 2009-2010(%)*
Enhancing granuloma	**63.3 %**	**54.05%**
Infarct	**4.0%**	**16.21%**
Mass	**4.6%**	**5.4%**
Calcification	**11.8%**	**5.4%**
Haemorrhage	**0.8%**	**5.4%**
Cerebral Atrophy	**5.9%**	**2.7%**
AVM	**0.8%**	**2.7%**
Gliosis	**0.5%**	**2.7%**
CVT	**Nil**	**2.7%**



Similar study in children was done in the year 2004 by Hussian etal. In their study the incidence of structural lesion was 68% which is almost close to our study. In their study also contrast enhancing granuloma was the most common lesion.

We have observed that patients with granulomatous lesions contrast enhancing lesions have more chance of their EEG being abnormal when compared to other patients with structural lesions.

Incidence of lateralizing EEG abnormality was also more in patients with granulomatous lesions.

LESION	*EEG ABNORMALITY*
Contrast enhancing granuloma	55%
Infarct	50%
Mass	50%
Others	22%

Distribution of granuloma was most commonly noted in parietal lobe 17/20 (85%) of which left side was more common than right with 30% more on the left compared to the right side. Overall 25 cases out of the 37 cases with structural lesion had their lesion in the parietal lobe (67.6%). So it shows that parietal lobe lesions are the most common cause of partial seizure.

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

1. Sex Incidence of Partial seizures are nearly equal with slight male preponderance.
2. The highest incidence in the age group of 10-19 years(26%)
3. 74% of patients with partial seizures showed lesions on CT scan.
4. Patients with postictal deficit have more chance of structural lesion.This study has shown 20 out of 23 patients who has postictal clinical signs or deficit had abnormal CT brain (87%).
5. Incidence of structural lesion in CT brain is more in patients who has an abnormal EEG, more so if EEG has lateralizing abnormality (82.35%).
6. Contrast enhancings granuloma was the most common cause for partial seizure (40%),particularly Tuberculoma of the brain was the most commonest cause, present in 34 %of patient, followed by CVA (12%).The most common age group in which tuberculoma was detected is 10-19 years.
7. Contrast enhancing granuloma were predominantly situated In the parietal lobe. Parietal lobe lesions were the most common cause of partial seizure.
8. EEG was most often abnormal in patients with contrast Enhancing granuloma (55%) when compared to other lesions(45%).

In 2 patients physical examination, CT Scan, EEG and other investigations did not reveal a causative factor. These should be further evaluated with MRI to rule out significant brain abnormalities and detect treatable causes if any.

It can be concluded that the probability of detecting a structural brain abnormality, treatable or otherwise, is very high .In patients with partial seizures, CT scan is indicated in all cases of partial seizures. Though newer imaging modalities like MRI may be more specific and sensitive, given the socioeconomic setup of our country ,CT scan still remains the most valuable tool in making etiological diagnosis of partial seizures

ANNEXURES

PROFORMA

ANNEXURE- I

PROFORMA

Name :

Age / Sex :

IP.No. :

Address :

Ward :

DOA : DOD:

Witness of seizure **Reliability**

H/o Seizure pattern:

Before the episode

- Recent illness (headache / fever)
- Unusual stress
- Medications
- Last alcohol intake
- Sleep deprivation
- Activity just before seizure

During the episode

- Time of day
- Type of seizure

  -simple partial

  -complex partial

  -Partial seizure with secondary generalization
- H/o Aura –present or not

  -If yes, Type of Aura- Auditory/ Sensory/ Olfactory

  visual
- Duration of Seizure :
- Side :
- Site :
- Status if any

  Ability to talk & comprehend

  Ability to recall events

Movements of eyes, face, arm,leg

Tongue bite, frothing

Bowel / bladder incontinence

Bodily injuries sustained

After event

- Confusion Duration
- Focal neurological deficits
- Headache
- Any other significant symptoms

SIGNIFICANT PAST HISTORY

- Diabetic : yes / no, if yes duration & treatment
- Hypertension / CAD/CKD
- Tuberculosis
- any others
- **alcohol intake** yes / no if yes duration, frequency, quantity, last intake
- smoking

FAMILY HISTORY OF SEIZURES

CLINICAL EXAMINATION

General exam:

Neurocutaneous markers

Vitals : BP Pulse RR Temp

CNS :

- higher functions
- motor system
- sensory system
- cranial nerves
- cerebellum:
- signs of meningial irritation

OTHER SYSTEMS –CVS/ RS/ ABDOMEN

COURSE DURING HOSPITAL STAY

INVESTIGATIONS :

Hematology - TC : DC: P L E B HB : ESR:

Biochemistry - sugar: urea: creatinine: Na : k : Ca

CSF analysis -

ECG -

Cx Ray -

ECHO -

CT BRAIN -

MRI BRAIN -

EEG -

TREATMENT

MASTER CHART

S.NO.	NAME	AGE /SEX	IP NO.	TYPES OF SEIZURE			SIDE OF THE SEIZURE		DEFICIT		EEG			CT SCAN BRAIN		
				SIMPLE	COM PLEX	2° GENERA LISATION	RIGHT	LEFT	PRESENT	AB SENT	ABNORMAL		NORMAL	ABNORMAL		NO RMAL
											GENER ALISED	LATERA LISED		RIGHT	LEFT	
1	KANNAN	7 / M	63478/09		Y		Y		Y				Y			y
2	UNNIKRISHNAN	23/ M	64321/09		Y			Y	Y		Y			Y		
3	SENNIYAPPAN	10/ M	67778/09	y			Y			Y	y					Y
4	AROKIARAJ	33/ M	68743/09		Y			Y	Y				Y	Y		
5	PARVATHY	27/ F	60004/09	Y			Y			Y	y					Y
6	JAYALAKSHMI	12/ F	71001/09			Y	Y		Y			y			Y	
7	MUREGESAN	45/ M	69967/09	Y				Y		Y			Y	Y		
8	NISHANTH	13/ M	68213/09	Y			Y			Y		y				Y
9	SANTHAMANI	57/ F	61241/09		Y		Y		Y			y			Y	
10	KALEED AHMED	26/ M	73421/09	Y				Y		Y			Y			Y
11	RATHINUM	33/ F	72178/09		Y			Y	Y				Y	Y		
12	FATHIMA	46/ F	66412/09		Y		Y		Y			y			Y	
13	KARTHIKA	9 / F	63901/09	Y			Y			Y			Y		Y	
14	MANIVEL	6 / M	65646/09		Y		Y		Y			y			Y	
15	SOUNDARIYA	22/ F	61716/09		Y			Y		Y			Y	Y		
16	PALANISAMY	51/ M	69898/09		Y		Y		Y		y				Y	
17	SHANU BAZEER	37/ M	70109/09	Y			Y			Y		y			y	
18	LAKSHMI	18/ F	71119/09		Y			Y	Y				Y	Y		
19	RAMALINGAM	69/ M	71919/09	Y				Y		Y			Y	Y		
20	POORNIMA	8 / F	69998/09		Y		Y		Y			y			Y	
21	MANI	28/ M	66768/09	Y				Y		Y		Y				Y
22	CHANDRASHEKAR	3 / M	65146/09		Y		Y		Y				Y			y

23	MYLSAMY	54/ M	69181/09			Y	Y		Y		y				Y	
24	RAJATHI	31/ F	67567/09	Y				Y		Y	y					Y
25	SHARATHA	29/ F	69191/09	Y			Y			Y			Y		Y	
26	MAYAWATI	45/ F	71016/09		Y			Y	Y				Y	Y		
27	PHILOMINA	17/ F	74178/10	Y			Y			Y			Y		Y	
28	NILOFUR	23/ F	74555/10		Y			Y	Y				Y	Y		
29	THILAGAVATHY	37/ F	75567/10	Y			Y			Y		Y				Y
30	NISHA	23/ F	76428/10		Y		Y		Y			y			Y	
31	SHAHUL HAMEED	7O/ M	76145/10	Y				Y		Y	y					Y
32	MUTHURAJA	16/ M	76981/10	Y			Y			Y			Y		Y	
33	SUGANYA	32/ F	77110/10		Y		Y		Y				Y			y
34	HASINA BEGUM	46/ F	76100/10		Y			Y	Y		y			Y		
35	SINGARAM	68/ M	78101/10		Y		Y			Y		y			Y	
36	VIMAL	15/ M	78367/10	Y				Y		Y			Y	Y		
37	SRIRAM	60/ M	78672/10	Y			Y			Y			Y		Y	
38	ROSHINI	13/ F	78918/10		Y		Y		Y			y			Y	
39	MOIDEEN	61/ M	78131/10		Y		Y		Y			y			Y	
40	DAMODHARAN	14/ M	79312/10	Y			Y			Y	y					Y
41	SURESH	58/ M	79718/10		Y			Y		Y			Y	Y		
42	GIRIJA	28/ F	79517/10		Y		Y		Y			y			Y	
43	RAJAN	63/ M	79532/10	Y				Y		Y			Y	Y		
44	KAMATCHI	18/ F	79813/10		Y		Y		Y		y				Y	
45	PUGAZHENDHI	70/ M	79103/10		Y		Y			Y			Y		Y	
46	RAVI	17/ M	79718/10		Y			Y	Y			y		Y		
47	MYLATHAL	64/ F	79412/10	Y			Y			Y			Y		y	
48	RAMAIYAH	19/ M	79618/10		Y			Y		Y		y		Y		
49	GIRIBALA	38/ F	80017/10		Y			Y		Y		Y				Y
50	SELVARAJ	17/ M	81321/10		Y			Y		Y			Y	Y		

CERTIFICATE OF CONSENT

ANNEXURE - II

CERTIFICATE OF CONSENT

I have invited to participate in research on study of 50 cases of focal seizures with CT Scan correlation. I understand that it will involve answering a detailed questionnaire, undergoing a thorough physical examination. I have informed that the risks are minimal and may include slight pain and redness at the site of needle prick. I am aware that there may be no benefit to me personally and that will not be compensated monetarily. I have provided with the name of the researcher who can be easily contacted using the number and address I was given for that person. I consent voluntarily to participate as a participant in this research and understand that I have the right to withdraw from the research at anytime without in anyway affecting my medical care.

Name of the participant:

Signature of the participant:

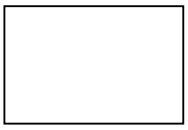
Date:

If illiterate, a witness must sign (if possible, this person should be selected by the participant and must have no connection to the research team)

I have witnessed the accurate reading of the consent form to the potential participant, translated to his/her mothertongue, and the individual

has had opportunity to ask questions. I confirm that the individual has given the consent freely.

Name of the witness **Thumb print of participant**



Signature of the witness:

Date:

I have accurately read or witnessed the accurate reading of the consent form to the potential participant, I confirm that the individual has given the consent freely.

Name of the researcher:

Signature of the researcher with date:

ABREVIATIONS AND ACRONYMS

SPS	-	Simple partial seizures
CPS	-	Complex partial seizures
TCS	-	Tonic -Clonic seizures
MTS	-	Mesial Temporal sclerosis
EEG	-	Electro EncephaloGram.
CSF	-	Cerebro Spinal Fluid.
CT	-	Computerized Tomogram.
MRI	-	Magnetic Resonance Imaging.
ILAE	-	International League Against Epilepsy
GTCS	-	Generalized Tonic- Clonic Seizure
CVT	-	Cortical Venous Thrombosis
CVA	-	Cerebro Vascular Accidents.
CP angle	-	Cerebello Pontine Angle.
NCC	-	Neurocysticercosis
AED	-	Anti Epileptic Drug.
SOL	-	Space Occupying Lesion

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Jerome Engel JR.,Timothy Pedley : Historical perspectives of Epilepsy – A Comprehensive Text book : 1998 : vol.I Chapter 3, page 15,

2. Annagers JF et al : Risk of recurrent seizure after an initial unprovoked seizure Epilepsia : 1986 : 27 : 43-50

3. S.Misra et al : CT Observation in partial seizure : Neurology India 42 ;24-27 : 1994.

4. Wagner A. : A clinical and epidemiological study of adult Patients with epilepsy. Acta Neurologica Scantinavia, Supp 1983 : 94 – 63-72.

5. Ottman. : Localisation of a gene for partial epilepsy –Nat. Genet 1995 : 10: 56- 60.

6. JMK Murthy : Clinical characteristics seizure spread patterns and prog.of seizures associated with single small cerebral calcific CT lesion. –1998, Seizure 153-57.

7. Adams and Victor's : Epilepsy and other seizure disorder,Principles of Neurology –7th edition, page 336.

8. Radhakrishnan et al : Pathology of TLE – Analysis of 100 Consecutive surgical specimens of patients with medially Refractory complex partial seizure. Neurology India : 1999 : 47 : 3: 196-201.

9. Joseph F. Hulihan : Focal EEG wave form abnormalities : – www Emedicine.com / neuro / topic

10. Gilmore PL : Correlation of EEG, CT and clinical findings –Study of 100 patients with focal delta activity –Arch of Neurology, 1981 : Jun 38 (6) ; 371-2.

11. Foldvary : Localising value of ictal EEG in focal epilepsy – Neurology 01: 57; 2022-28.

12. Wahringer Gurtal : Focal features in patients with Idiopathic Generalized seizure –18-20. Vienna Acta Epilepsy Res : 02 : Aug 50(3) – 293 – 30.

13. Devinsy et al : Clinical and EEG features of simple partial seizure Neurology : 1988 ; 38, 1347-52.

14. John Hughe's and Jhon I. Finn : Focal Seizure and EEG :Prognostic consideration, www. ecns web. com. / journal / ecns

15. Ropposelli : 3TPA MRI is more sensitive than 1.5T for Identifying focal lesions. Applied Neurology Nov : 05

16. Ramesh Bahti et al : Study of CT and EEG in patients with Partial epilepsy. – Indian academy of clinical medicine vol. 4 no. 1 ; Jan-Mar-03.

17. M.Artena et al : Clinical and EEG features and patients with wicket rhythm misdiagnosed as epilepsy Neurology 64 : 187 2005.

18. Hussein et al : Cranial computerized Tomography in partial Motor seizures, Indian Journal of Paediatrics 2004 : 71 : 641-44.

19. Chopra et al. : CT scan study in patients with partial seizures; Journal of Neurological sciences 107 : 40-49.

20. Vedhantham Rajasekar, Mattew Chandy et al : Solitary Cysticercus granuloma – disappearing lesions.

21. Misra S. et al : CT observation in partial seizures. Neurology India, 1994: 42 : 24-27.

22. F.J. Rugg, Gunn et al : Whole brain T2 Mapping demonstrates occult abnormalities in focal epilepsy. Neurology 2005 : 64 : 318- 325

.

23. Spencer SS. et al : Diagnosis of Mesial Temporal lobe seizure onset :relative specificity and sensitivity of quantitative MRI –Neurology 1993 ;43 ; 2117-2124.

24. Jerome Engel JR, Timothy Pedley : Positron Emission Technology :Epilepsy – comprehensive Text book 1998 Vol I : Page 963.

25. Handforth et al. : Markedly increased MST metabolism in the case with PLEDS further evidence that PLEDS are a manifestation of partial status epilepticus– Epilepsia 1994: 35 :876-881.

26. Mathias J Koepp et al : Imaging structure & function in Refractory focal epilepsy – Lancet Neurology Vol 4(1) Jan 05 –\\ 42-53.

27. LiLM, Fish DR, JNNP : High resolution MRI in adults with Partial or 2 generalised epilepsy attending a tertiary referral Unit 1995 : 59 ; 384-87

28. Thissu et al : Acute Symptomatic seizure due to single CT brain- how long to treat with AED –Neurology India 02:50:2;141-44.

29. Daniel H. Lowenstein : Seizures and epilepsy –Harrison's Principles of Internal Medicine 15th ed. Vol II, 2363 – 2366.

30. Dhanraj et al. : Gabapentin in refractory partial seizures –Neurology India : 1998 : 46 : 2 ; 106-109.

31. Sethi et al, : Gabapentin and Lamotrigine are indicated in patients with partial seizures refractory to carbamazepin Neurology India 62 : 50 ; 3: 359- 63.

32. Singhvi et al. : Profile of intractable epilepsy in a tertiary referral centre Neurology India, 48 : 4 : 351 – 56.

33. Wiebe S. et al : Controlled trial of Surgery for Temporal lobe epilepsy–NEJM, 01 : 345 ; 311-18.

34. Loyllice : Seizure outcome after epilepsy surgery in children and adolescent, Ann of Neurology : 1998 ; 44; 740-742,748.

35. Daniel et al. : Epilepsy surgery ; Over view of 40 years of experience :Neurology India, 1999; 47 : 2 ; 98-103.